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ON

GENETIC CONTROL OF THE IMMUNE RESPONSE

MAY 1, 1979

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
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SELECTED ABSTRACTS
ON

GENETIC CONTROL OF THE IMMUNE RESPONSE

A service of the International Cancer Research Data Bank (ICRDB) Program of the National Cancer Institute

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INTRODUCTION

This ONCOLOGY OVERVIEW on The Genetic Control of the Immune Response covers the publication years from approximately 1970 to the recent entries in the Cancerline data bases. The strategy used for the online data base search is available on request from the CIDAC office and can be entered at any MEDLARS/MEDLINE terminal system in order to obtain continuing updates in this area.

The scope of this overview covers the genetic basis of the control of the immune response in both animals and humans, the structure and expression of the Major Histocompatibility Complex (MHC) products and the physical association of MHC products with other types of antigens. Natural killer cell activity and genetic resistance in rodents is also covered. The association of certain HL-A haplotypes and specific human cancers is included.

This overview was prepared by Harvey Cantor (Editor) at the Sidney Farber Cancer Institute, Boston, Massachusetts and Barbara Hindman of the CIDAC for Virology, Immunology and Biology, The Franklin Institute Research Laboratories.

EDITORIAL COMMENTARY

For the past ten years a large body of experiments has indicated that the small cluster of genes located on chromosome 17 of the mouse controls the duration, intensity, and type of immune responses to a wide variety of antigens. This region, spanning the two transplantation loci "H2-D" and "H2-K," is known as the Major Histocompatability Complex (MHC), because these genes code for antigens which induce the strongest homograft reactions. A cluster of genes that map between K and D has been termed the "Ir(immune response)-gene region;" throughout this region are genes that encode for surface components (called Ia molecules) that are expressed primarily on lymphocytes and macrophages. There is increasing evidence that genes within the HL-A complex in man may code for analogous lymphocyte-surface components.

The precise mechanism of Ir gene control is not well understood. There is increasing evidence that freely circulating T-lymphocytes are equipped with receptors that bind to both Ia molecules and K/D region products that are associated with foreign antigens such as a virus. A central function of these MHC gene products may be to activate two sublines of T cells that are genetically programmed to induce either humoral or cellular immune reactions: T-helper cells respond to antigen associated with I-region products, T-cytotoxic cells react to antigen associated with H-2K/D gene products. The selective altering of either screening system by foreign or altered H-2K/D or I-region products thus activates a series of immune reactions already programmed in one or another T-cell set. This reaction, if successful, results in clonal expansion and immune elimination of cells bearing "non-self" determinants.

In certain situations, lack of a detectable immune response to certain antigens may be due to inability of these antigens to bind to Ia molecules on macrophages. In other cases, macrophage processing results in antigen-Ia complexes which selectively induce antigen-specific suppressor T cells and no detectable immune response.

Whether tumor express antigens that fail to induce T-cell responses or selectively induce suppressive activity is now a crucial question for both understanding and manipulating the immune system in the face of tumor growth. The following abstracts summarize experimental information indicating that: 1) the cellular basis of Ir gene control may reflect, in part, expression of different sets of Ia molecules and selective or preferential generation of suppressive T cells by certain antigens (including tumor associated determinants); 2) T cells that are equipped to kill cells (including certain tumors) recognize a foreign antigen in association with MHC products; 3) the MHC region also controls the expression of cytotoxic behavior by lymphocytes (called "natural killer" cells) that spontaneously damage or destroy tumor cells; and 4) certain HL-A linked genes in man may be associated with increased incidence of certain tumors.

I. GENETIC AND MOLECULAR BASIS OF ANTIGEN RECOGNITION BY CYTOTOXIC T CELLS

A. Structure and Expression of Major Histocompatibility Antigens on Normal and Neoplastic Cells

1. STRUCTURE AND BIOLOGICAL ACTIVITY OF H-2 ANTIGENS. (PP. 667-676)

Cunningham BA, Henning R, Schrader JW, Milner RJ, Reske K, Edelman GM
The Role of Products of the Histocompatibility Gene Complex in Immune Responses. Katz DH, Benjacerraf B, ed. New York, Academic Press, 1976.

Current knowledge of the structure and biological activity of the H-2 antigens is reviewed. H-2 antigens consist of two polypeptide chains: a heavy one (molecular wt 50,000), which bears antigenic specificity and a carbohydrate, and a light one (molecular wt 12,000), which has been shown to be homologous to the constant regions of immunoglobulin light and heavy chains. The detergent solubilized molecule in soln is composed of two disulfide-linked heavy chains and two noncovalently associated light chains. On the cell surface, the H-2 antigen is primarily a monomer, but dimers or disulfide-linked dimers may exist, at least transiently. Comparisons of the partial amino acid sequences of H-2Kb, H-2Kd, H-2Kk, H-2Db, and H-2Dd gene products support the hypothesis that the K and D loci evolved by gene duplication. H-2 antigens are involved in cell-mediated lysis of allogenic and xenogenic cells as well as the lysis of chemically modified and virally infected syngeneic cells and syngeneic tumor cells. Several lines of evidence indicate that H-2 antigens can be physically associated with viral antigens on the cell. Further, H-2 antigens and viral antigens can co-cap and co-patch on the cell surface. A suggested function for H-2 antigens is that of serving as adaptors that combine with foreign antigens to form hybrid antigens, which are recognized by cytotoxic T lymphocytes. (19 refs)

2. THE HISTOCOMPATIBILITY SYSTEMS.

Snell GD
Jackson Lab., Bar Harbor, Me.
Transplant Proc; 3(3):1133-1138 1971

Cell membrane bound alloantigens are currently demonstrated by three methods: (1) the use of tumor or skin or, less commonly, marrow or other tissue transplants, in appropriately designated inbred strains or segregating generations; (2) blood typing; (3) study of alloantigens of nucleated cells of the blood or lymphoid organs by a cytotoxic technique requiring alloantibody and complement. These three methods are used to classify histocompatibility loci; the question whether or not the method of classification used results in distinct locus groupings is discussed. The so-called histocompatibility genes are those which determine the acceptance or rejection of transplants; the end products of these genes are alloantigens localized in the cell membrane and thus are vulnerable to the immune defenses of the graft recipient. These genes may be part of a family of gene loci either directly or indirectly concerned with histocompatibility. This larger group of loci is referred to as "cell membrane alloantigen determining loci" (CMAD loci). Cell membrane bound alloantigens can be demonstrated by three methods, and antigens are accordingly classified as to their method of identification. One standardized system for classifying and naming CMAD loci is detailed. Evidence bearing on the question of the distinctiveness of the CMAD loci in this system is discussed. Evidently, histocompatibility loci show great diversity with respect both to tissue distribution of their end products and to different arrangements of the end products on the cell surface. Loci demonstrated through an effect on histocompatibility (H-loci, in the classification system) may represent a rather distinct group. Methods used to demonstrate anti-H antibodies by hemagglutination may not be entirely valid. It is also suggested that the glycoprotein coating found over cell membranes may mask H-antigens under some conditions.

3. TUMOR-ASSOCIATED ANTIGENS IN H-2 HEMIZYGOUS ISOANTIGENIC VARIANTS OF A SOMATIC CELL HYBRID, DERIVED FROM THE FUSION OF A 3-METHYLCHOLANTHRENE-INDUCED SARCOMA AND A MAMMARY CARCINOMA.

Klein G
Department of Tumor Biology, Karolinska Institutet, S 104 01 Stockholm 60, Sweden
J Natl Cancer Inst; 58(2):383-386 1977

To check the suggestion that 3-methylcholanthrene-induced, sarcoma-associated, tumor-specific transplantation antigens (TSTA) could be modified H-2 antigens, reciprocal isoantigenic variants derived from TA3Ha/MSWBS hybrid cells were examined. This hybrid was produced by the fusion of the TA3Ha ascites carcinoma (H-2**a) and the 3-methylcholanthrene-induced MSWBS ascites sarcoma (H-2**s). MSWBS expresses a strong TSTA capable of inducing a rejection reaction in the syngeneic A SW host. The genetic determinants of the H-2 complex are known to be localized on chromosome No 17. TA3Ha contributes two normal, telocentric chromosomes No 17 to the hybrid. In contrast, both chromosomes No 17 of MSWBS are localized on readily identifiable translocations (17/1 and 17/M1). We have previously shown that the chromosomes No 17 of one parental strain or the other (but not both) can be eliminated from the hybrid upon selective passage in the opposite parental strain. The present studies showed that the two strain A-compatible variants that have lost the sarcoma-derived 17-chromosomes still contained the same TSTA as the two reciprocal strain A SW-compatible variants that have lost the mammary carcinoma-derived H-2 chromosomes. These findings argue against the possibility that methylcholanthrene-induced TSTA is a modified form of H-2 or that its structural determinant(s) is localized on chromosome 17. (Author Abstract)

4. PARENTAL VARIANTS.

Klein E
Karolinska Inst., Stockholm, Sweden
Transplant Proc; 3(3):1167-1171 1971

A study of cellular variation in terms of gene products was carried out. F1 crosses were obtained between strain A mice and the three coisogenic lines ASW, ABY, and ACA. The resultant mice, differing genetically at the H-2 locus, were then subjected to methylcholanthrene injections to stimulate tumor growth. Once sarcomas were formed, transplantation was accomplished into the filial mice to determine homograft reaction. In mice which developed tumors, tumors were highly specific for genotype, the transplantation behavior being irreversible once selected; in addition, when transplant was carried out over successive generations, each variant subline was more specific as compared to the original tumor. Further information was obtained by immunogenicity (induction of antibody) tests and reactivity with specific antisera. It was found that the original tumor was asymmetric with regard to the expression of the two parental specificities, the cells apparently possessing a higher amount of one antigen than another. Additional genetic crosses and tumor transplantation techniques demonstrated that the loci controlling various types of antigen could be lost separately or together depending on their position relative to the centromere, and that some of these loci were not always necessary for cell viability since antigens could be regained over a period of time in the absence of loci. This regaining of antigens probably occurs because the cell surface of the tumor retains the antigens in reduced concentrations, these antigens being enhanced on stimulation. However, it is concluded that at least one set of H-2 antigens is needed for cellular viability of the tumor.

5. ANTIGENIC CHARACTERISTICS OF MURINE LEUKEMIA INDUCED BY CHEMICAL CARCINOGENS.

Colnaghi MI, Della Porta G
Natl. Cancer Inst., Milan, Italy
Bibl Haematol; (39):595-602 1973

The antigenicity of thymic lymphosarcomas was characterized in male and female mice of C3Hf, C57BL, and SWR strains. Ten day old animals received five 1 mg/g ip injections of urethane (1 injection/day) on alternate days to produce thymic lymphosarcomas. Lymphomas were kept by transplantation in preirradiated syngeneic hosts and were at no more than the fifth passage when used. Mice were immunized using 5-6 inocula of a cell suspension prepared from either a single or multiple (2-3) tumors. Irradiated (10,000 r) or blocked (mytomycin 250 microg/10 x 106 cells) tumor cells were used for syngeneic immunization, while allogenic immunizations used both treated and untreated cells. Membrane fluorescence, trypan blue exclusion and 51Cr-release in vitro tests were used to measure immune response, considering only cell surface antigenicity. Sera were always syngeneic to the target cell (no alloantigens) and were used undiluted, after inactivation. Syngeneic immunizations always yielded a negative cytotoxic or fluorescence index; allogeneic immunizations gave positive indices, as did blocked tumor cells. An effective immunity from humoral and cell-mediated responses was obtained when immunized and immunizing strains differed in the H-2 alleles. To demonstrate the cross-reacting tumor-specific antigenicity, C57BL anti K36 antiserum absorptions were conducted. Several lymphomas completely absorbed the cytotoxicity activity of the antiserum. The results show that the urethane-induced lymphomas possess the Gross virus-associated antigenicity. Further investigation of the Gross cell surface antigen in the urethane-induced lymphomas and the antigen detected by the antisera from allogeneic immunizations is suggested.

6. PROPERTIES OF RETICULUM CELL SARCOMAS IN SJL/J MICE. V. NATURE OF RETICULUM CELL SARCOMAS SURFACE ANTIGEN WHICH INDUCES PROLIFERATION OF NORMAL SJL/J T CELLS.

Ponzio NM, Daivd CS, Shreffler DC, Thorbecke GJ
Dept. Microbiology - Immunology, Northwestern Univ.
Sch. Medicine, Chicago, IL 60611
J Exp Med; 146(1):132-145 1977

Studies of reticulum cell sarcoma (RCS) tumors in SJL/J mice indicate that spontaneous tumors that arise in older mice possess the capacity to induce the vigorous proliferative responses in syngeneic T-lymphocytes that are characteristic of the transplantable RCS lines. Analysis of cell surface antigens revealed the presence of Ia determinants on gradient-purified transplantable RCS tumor cells; however, these cells did not express Thy 1.2, Ig, or any of the viral proteins that were tested for by specific antisera. Pretreatment of RCS cells with anti-Ia sera and complement-deleted cells that were stimulatory for syngeneic T lymphocytes, and the addition of anti-Ia sera directly to cultures, blocked the proliferative responses at the stimulator (RCS) cell level. Lymph node cells from H-2s strains other than SJL/J, including A.SW and B10.S, also gave proliferative responses to RCS cells, although lower in magnitude. A requirement on the part of responding cells for identity with RCS cells at the Ir region was indicated by the finding that A.Th but not A.TL lymph node cells responded to RCS. It is concluded that RCS cells stimulate Ir-region identical T-cells (without evidence of presentation) through a modification in the expression of Ia antigens on the surface of the tumor cells. (51 Refs)

7. FURTHER EVIDENCE FOR DEREPRESSION OF H-2 AND IA-LIKE SPECIFICITIES OF FOREIGN HAPLOTYPES IN MOUSE TUMOUR CELL LINES.

Garrido F, Festenstein H, Schirrmacher V
Tissue Immunology Unit, London Hosp. Medical Coll.,
Turner St., London E1 2AD, England
Nature (Lond); 261(5562):705-707 1976

According to the derepression hypothesis under investigation, allelic differences in histocompatibility antigens are the products of closely linked genes, most of which are repressed in normal circumstances. A virus or other agent could interfere with this repression system and cause derepression of certain genes and/or repression of others, resulting in the appearance of allogeneic and/or the loss of syngeneic specificities. The following tumor cells were propagated in ascites form in syngeneic recipients: (a) cells from a spontaneous u-positive mouse lymphoma (SL2) expressing in addition to the H-2 specificities of the host's cells, alloantigens (H-2 and Ia) apparently of foreign haplotypes; and (b) a chemically induced lymphosarcoma (Gardner) not expressing any of the private H-2 specificities of the host's cells. They were taken from the peritoneal cavity and subjected to a direct complement-dependent microcytotoxicity test and a postlabeling assay that measures the uptake of ^{14}C-thymidine. SL2 lymphoma cells reacted strongly with all antisera against the private and public specificities of the host. Antiserum against the private H-2K specificity (H-2K.23) did not react with the Gardner lymphoma but did react with normal lymphocytes from the host, which indicates the loss of certain H-2 specificities. Ia specificities seemed to be expressed on one of the Thy 1.2 (alloantisera) positive tumors. The cytotoxic antitumor activity of anti-Ia sera could be absorbed with spleen cells from mice of foreign H-2 haplotypes, but not with spleen cells from H-2 identical mice or from serum donor strains. The data obtained support the derepression hypothesis, although the possibility of an activated endogenous virus directly creating new H-2-like determinants on the tumor cell surface cannot be excluded. The extra specificities were serologically indistinguishable from major histocompatibility complex (H-2 and Ia) specificities.

8. CHANGES IN HL-A ANTIGEN PROFILES ON SV40-TRANSFORMED HUMAN FIBROBLASTS.

Pellegrino MA, Ferrone S, Brautbar C, Hayflick L
Scripps Clinic and Res. Foundation, La Jolla, CA 92037
Exp Cell Res; 97(2):340-345 1976

The histocompatibility antigen profile of human fibroblasts transformed with simian virus 40 (SV40) was investigated by determining their ability to specifically absorb HL-A alloantisera. All four human adult skin fibroblast lines acquired, after SV40 transformation, the ability to absorb the

HL-A alloantiserum VICTOR directed against the specificities HL-A5, W5. On the contrary, none of six embryonic fibroblast lines showed any qualitative or quantitative change of their HL-A antigenic profile. Similarly, murine, monkey and hamster cells could not absorb the activity of the HL-A alloantiserum VICTOR after SV40 transformation. It is suggested that the "new HL-A antigen" specificity on human fibroblasts after SV40 transformation may be due to either a cross-reaction between HL-A specificities and antigenic structures present on glycoprotein(s) coded by the viral genome and expressed on the cell, or to a change in the regulating mechanism of the expression of histocompatibility antigens on the cell surface, should the genetic information for all the HL-A specificities be present in the cell genome.

9. A NEW SEROLOGICALLY DEFINED LOCUS, QA-1, IN THE TLA-REGION OF THE MOUSE.

Stanton TH, Boyse EA
Memorial Sloan-Kettering Cancer Center, New York, New York 10021
Immunogenetics; 3(6):525-531 1976

A new cell-surface antigen, specified by a gene between H-2D and TLA is described. The provisional notation Qa-1 is suggested for the locus determining this newly recognized cell surface component. Qa-1 is distinguished from known TL antigens by the following two criteria. Its expression is not confined to thymocytes - it occurs on lymph node cells (LNC) also; and the phenotypes of the new congenic recombinant strains B6 K1 and B6 K2, derived from H-2D/TLA crossovers, are Qa-1**+Qa-2**-TL**- and Qa-1**+Qa-2**+TL**-. Qa-1 antigen is defined by reaction of the standard TL typing serum, (B6 x A-TLA**b)F1 anti-A strain leukemia ASL1, with lymph node cells (LNC) in the cytotoxicity assay. Qa-1 antigen evidently is expressed, at least, on a subpopulation of T cells as well as on thymocytes. The gene order is H-2D, Qa-1, Qa-2, TLA. (Author Abstract)

B. Recognition of Viruses and Other Antigens in Association with Major Histocompatibility Products

10. H-2 REGION PRODUCT AS DETERMINANT IN IMMUNE CYTOLYSIS OF SYNGENEIC TUMOR CELLS BY ANTI-MSV T LYMPHOCYTES.

Gomard E, Duprez V, Henin Y, Levy JP
Laboratoire d'Immunologie et Virologie des Tumeurs, (Inserm U. 136), Service d'Hematologie Hopital Cochin, 27 rue Faubourg Saint Jacques, 75014 Paris, France
Nature (Lond); 260(5553):707-709 1976

Experiments to determine the role of the H-2 antigens in immune cytolysis of syngeneic tumor cells by anti-murine sarcoma virus (MSV) cytotoxic T lymphocytes (CTL) are reported. Anti-MSV CTL were derived from the spleens of the following strains 10-16 d after virus inocn.: BALB/c (H-2d), B10.D2 (H-2d), BALB.B (H-2b), C57BL/6 (H-2b), and (BALB/c x C57BL/6)F1 (H-2b/d). Tumors used were YC8 and LSTRA in BALB/c (C) and MBL2 and FBL3 in C57BL/6 (B6). B6 anti-MSV CTL were strongly cytolytic for B6 targets and only weakly cytolytic for C targets, while the reverse was true for C anti-MSV CTL. (B6 x C)F1 CTL, which possess both H-2b and H-2d, were fully effective against both types of targets. The H-2 specificities are determinant in the reaction. Hybrids were immunized against the virus or by MBL2 or FBL3 (B6) or LSTRA (c) tumor cells. Hybrids immunized against the virus produced CTL which reacted with both MBL2 and YC8, but hybrids immunized against C or B6 tumor cells produced CTL which reacted only with the immunizing cells or antigenically related cells of the same line. H-2 compatibility between CTL and the target was not sufficient to permit immune cytolysis. Anti-MSV CTL may recognize only one structure on the target cell surface - an antigen which is different according to the H-2 of the host. To demonstrate a close topographical relationship between H-2 and MSV antigens, C anti-B6 and B6 anti-C antisera were prepared by repeated inocn of normal spleen cells. These sera were used either unabsorbed or after absorption in vivo oncogenic spleen cells to provide H-2 and nonH-2 typing sera. C anti-B6 absorbed on BALB.B provided a non H-2 typing serum, while the same sera absorbed on B10/D2 provided on H-2 typing serum. Unabsorbed and anti-H-2 typing serum blocked the activity of anti-MSV CTL, whereas nonH-2 typing sera were devoid of blocking activity. Furthermore, a BALB/c anti-BALB.B typing serum which reacted only with H-2 was fully blocking. Anti-Thy1-2 (AKR anti C3H/He) incubated with Thy1-2(+) MBL2 lymphoma cells did not inhibit their cytolysis by anti-MSV CTL. In addition, unrelated anti-H-2 (e.g., B6 anti-C on B6 cells attacked by B6 anti-MSV CTL) were not blocking. Anti-H-2 have a definite and specific blocking activity for anti-MSV CTL and a close topographical relationship exists between H-2 and MSV antigens. (14 refs)

11. VIRUS-IMMUNE CYTOTOXIC T CELLS ARE SENSITIZED BY VIRUS SPECIFICALLY ALTERED STRUCTURES CODED FOR IN H-2K OR H-2D: A BIOLOGICAL ROLE FOR MAJOR HISTOCOMPATIBILITY ANTIGENS. (PP. 387-389)

Zinkernagel RM, Doherty PC
Immune Reactivity of Lymphocytes Development, Expression, and Control. Feldman M, Globerson A, ed. New York, Plenum Press, Advances in Experimental Medicine and Biology; vol. 66, 1976.

T cell mediated lysis of macrophages, tumor cells, or fibroblasts infected with lymphocytic choriomeningitis (LCM) virus occurs only if lymphocyte and target are compatible at the H-2 gene complex. Experiments with ectromelia and paramyxovirus infection indicate that this may be a general phenomenon as these viruses differ considerably in their effects at the molecular level. Mapping studies have indicated that compatibility at either the K or the D region of one H-2 haplotype is sufficient for this lytic interaction. The Ir-Ss regions are not involved in either a primary or secondary role. The phenomenon appears to be associated with identity at the serologically-defined H-2 private specificities. An essential implication of these findings is that the surveillance T cell is programmed to monitor H antigen structural integrity. (13 refs)

12. RELATIONSHIPS BETWEEN H-2 AND VIRAL ANTIGENS IN MURINE ONCORNAVIRUS-INDUCED TUMOURS.

Gomard E, Duprez V, Henin Y, Levy JP
Batiment Gustave Roussy, 7eme etage, 27 rue du Faubourg Saint Jacques, F75014 Parks, France
J Immunogenet; 4(1):35-45 1977

Cytotoxic T lymphocytes (CTL) from murine sarcoma virus (MSV) or Friend leukemia virus (FLV)-inoculated mice lysed syngeneic FMRGi+ (Friend, Moloney, Rauscher, or Graffi) lymphoma cells much more efficiently than allogeneic FMRGi+ lymphoma cells. The cytolysis of various H-2 antigen-different ^{51}Cr lymphomas was composed by the CTL from several inbred and congenic lines differing at H-2 and by competition experiments using unlabeled cells. The results indicate that this phenomenon is due to an H-2 barrier. H-2b/H-2d hybrid-anti-MSV-CTL immunized by H-2b, H-2d, or H-2b/H-2d tumors lysed only FMRGi+ lymphomas of the same H-2-identical competitive cells. This indicates that H-2 antigens are directly involved in the interaction between tumor cells and immune CTL that probably react with an H-2 modified antigen on the tumor cell surface. The use of CTL from intra-H-2 recombinant lines shows that H-2D and probably H-2K molecules are involved, but they vary according to the tumor cells. It could not be determined if the I region plays a role in the CTL/tumor cell interaction. The possible effects of Ia antigens due to I-A and I-B subregions cannot be excluded in the combinations tested. It is concluded that restriction of the cytolysis of tumor cells by anti-MSV-CTL and anti-FLV-CTL is related to H-2 antigens. These antigens could play a major role in immunosurveillance against oncogenic and nononcogenic viruses and against viral tumors. (23 Refs)

13. MECHANISMS OF THE H-2 EFFECT ON VIRAL LEUKEMOGENESIS.

Bubbers JE, Blank KJ, Freedman HA, Lilly F
Dept. Genetics, Albert Einstein Coll. Medicine, Bronx, NY 10461
Scand J Immunol; 6(5):533-534 1977

Studies of the effect of the H-2 haplotype on the immune response of congenic BALB mice to Friend murine leukemia virus (FV)-induced tumors are reviewed. The results support the notion that two distinct but interacting mechanisms, controlled by loci mapping within the H-2 complex, influence FV disease. One mechanism, controlled by a gene mapping in or close to H-2D, influences the capacity of the H-2D gene product to form molecular complexes with FV molecules in the plasma membrane of infected cells. Complex formation appears to provide a target antigen for syngeneic cytotoxic T lymphocytes, to cause cocapping of FV and H-2D antigens, to permit the selective inclusion of H-2D molecules into progeny Friend virions, to influence the long-term maintenance of virus production in vitro, and, in conjunction with the second mechanism, to stimulate the generation of cytotoxic T-lymphocytes. This second mechanism is controlled by a gene in the H-2K or H-2I region, and, in the presence of an H-2/FV molecular complex immunogen, influences the generation of H-2 restricted cytotoxic T lymphocytes and the rate of rejection of syngeneic FV-induced tumor cells. (16 Refs)

14. IMMUNE MECHANISMS IN LEUKEMIA: ROLE OF THE IA ANTIGENS.

Lukasewycz OA, Frelinger JA
Dept. Medical Microbiology and Immunology, Univ. Minnesota Sch. Medicine, Duluth, MN 55812
J Exp Med; 145(4):1077-1081 1977

The I (immune response) region of the mouse major histocompatibility complex codes for a series of membrane alloantigens (Ia) that are expressed on at least a subset of T lymphocytes. The role of Ia+ T-lymphocytes in protecting immunosuppressed (Cytoxan, 250 mg/kg) C58 mice against challenge with syngeneic line Ib leukemia cells (10^3) was investigated. The protective capacity of nylon wool-purified T cells was completely abrogated by treatment with complement and anti-Ia antisera, which resulted in the removal of the small population (10%-15%) of cells possessing the Ia determinants coded by the I-A, I-B, and I-J regions of the H-2 gene complex. This suggests that Ia+ T cells have a significant role in tumor immunity. (23 Refs)

15. INFLUENCE OF H-2 COMPLEX ON SUSCEPTIBILITY TO INFECTION BY MURINE LEUKEMIA VIRUS.

Tucker HS, Weens J, Tsichlis P, Schwartz RS, Khiroya R, Donnelly J
Hematology Service, New England Medical Center Hosp., Boston, MA 02111
J Immunol; 118(4):1239-1243 1977

The effect of the H-2 complex on the early phase of infection by murine leukemia virus (B-tropic MuLV) was studied. Several strains of congenic resistant mice were used. In mice differing only in H-2 haplotype, a gene(s) within the H-2 complex was found that determined a high (H-2k, H-2d, H-2a) or low virus titer (H-2b, H-2q). For example, 10 wk after infection with virus (200 plaque-forming units), B10 mice had significantly lower titers of infectious virus in their spleens than B10.A mice. (B10 X B10.A)F1 mice were phenotypically like the B10.A parent; thus, susceptibility to high virus titers was inherited as a dominant trait. Kinetics experiments with strains B10 and B10.A showed that no infectious virus was detectable in both until 4 wk after infection. From 4 to 6 wk the titers of infectious virus rose, but from 6 to 10 wk, the titers in B10 mice declined progressively. This suggests that there is a mechanism in B10, lacking in B10.A, that restricts viral replication after infection. Antibody to viral envelope antigen titers differed significantly between the two strains, but the antibody did not appear to be involved in virus elimination. Genes outside the H-2 complex influenced MuLV titers after infection as well. (20 Refs)

16. T-CELL-MEDIATED CYTOTOXICITY AGAINST HERPES SIMPLEX VIRUS-INFECTED TARGET CELLS.

Pfizenmaier K, Starzinski-Powitz A, Rollinghoff M, Falke D, Wagner H
Institut fur Medizinische Mikrobiologie der Johannes Gutenberg-Universitat, 65 Mainz, Hochhaus am Augustplatz, W. Germany
Nature (Lond); 265(5595):630-632 1977

A different experimental approach was used to show that T lymphocytes of mice undergoing a local herpes simplex virus (HSV) infection, when transferred in vitro, differentiate into effective cytotoxic T lymphocytes (CTL). These CTL are highly cytotoxic for HSV-infected target cells. About 1-5 x 10^5 plaque-forming units (PFU) of HSV type 1 strain Lennette were injected into the hind footpad of CBA/J mice. After 3 days the draining lymph nodes (LN) were isolated and teased, and single-cell suspensions were prepared and cultured for an additional 72 hr at 37 C. LN cells were then tested for the presence of virus-specific CTL in a 6-hr cytotoxicity assay using thioglycollate-induced ^{51}Cr-labeled, HSV-infected CBA macrophages as targets. The syngeneic target cells were infected in vitro with 2-5 PFU of HSV/cell 16 hr before the assay. LN cells draining the site of a local HSV infection in vivo, when transferred in vitro, differentiated into cytotoxic lymphocytes with strong activity toward HSV-infected syngeneic macrophages. LN cells draining a site injected only with culture medium (control) did not develop cytolytic activity. Data strongly suggested that these cells were T-cells. In most experiments, CTL generated lysed noninfected syngeneic targets only to a limited degree, and virus-specific cytotoxicity was tested in an experimental system in which the time of contact between CTL and HSV-infected target cells could be reduced to less than 1 hr. Results indicated that this contact time was sufficient for strong lysis of targets. Also, cytolytic activity was restricted to HSV-infected targets; no lysis of normal targets was found. Further studies showed that the lytic activity is restricted to recognition of HSV antigens in the context of syngeneic histocompatibility antigens (H-2 restriction). HSV-induced CTL may be involved in controlling the in vivo spread of HSV infection, and they may be cytotoxic toward infected targets well before viral progeny are produced. (10 refs)

17. DOES THE APPARENT H-2 COMPATIBILITY REQUIREMENT FOR VIRUS- SPECIFIC T CELL-MEDIATED CYTOLYSIS REFLECT T CELL SPECIFICITY FOR "ALTERED SELF" OR PHYSIOLOGICAL INTERACTION MECHANISMS? (PP. 203-211)

Zinkernagel RM, Doherty PC
The Role of Products of the Histocompatibility Gene Complex in Immune Responses. Katz DH, Benacerraf B, ed. New York, Academic Press, Inc, 1976.

The apparent H-2 compatibility requirement for virus-specific T cell- mediated cytolysis is studied as a reflection of T cell specificity for altered self or physiological interaction mechanisms. The virus-specific T cells lysed infected targets only when they were compatible at the D or K region of H-2. Results with H-2K mutant Hz170 and Hz1 mice indicated that the cistron coding for the structure in the K region was defined by the two mutants. The T cells were thought to be specific for virus altered self markers coded in the D or K region of H-2. Because neither the antigenic entity of altered self nor the hypothetical interaction structure for like self has been biochemically defined, all arguments are based on circumstantial evidence derived from biological experiments. It is therefore, concluded that it is premature to exclude either of these mechanisms as an explanation for the phenomenon. (24 refs)

18. ROLE OF THE MURINE MAJOR HISTOCOMPATIBILITY COMPLEX IN THE SPECIFICITY OF IN VITRO T-CELL-MEDIATED LYMPHOLYSIS AGAINST CHEMICALLY-MODIFIED AUTOLOGOUS LYMPHOCYTES.

Shearer GM, Rehn TG, Schmitt-Verhulst AM
Natl. Cancer Inst., Natl. Inst. Health, Bethesda, MD 20014
Transplant Rev; 29:222-248 1976

The genetic and immunologic parameters associated with in vitro T-cell-mediated lympholysis (CML) to tri-nitrophenyl (TNP)-modified and N-(3-nitro-4-hydroxy-5-iodophenylacetyl)-beta-alanylglycylglycyl-(N-) modified autologous lymphocytes are reviewed. Efficient generation of CML reactions to viral infected cells in vivo and to chemically modified cells in vitro requires H-2 homology between target cells and the cells involved in the sensitization phase. The H-2 homology may be explained by one of at least three mechanisms: (a) that the CML is essentially hapten-specific, but that some type of cell-to-cell interaction is required involving intra-H-2 identity between responding and stimulating cells for sensitization to occur, and between effector and target cells for efficient lysis to occur; (b) the responding lymphocytes and their cytotoxic descendents recognize the chemical agent or virus and unaltered cell surface major histocompatibility H-2 complex (MHC) products as a single recognition unit; and (c) the chemicals or viruses act as modifying agents, which immunologically alter cell surface structures controlled by genes mapping in or near the serologically defined MHC regions. In the second possibility, at least some of the chemical or viral structures may be situated on the cell surface in close proximity to the relevant H-2 region products because anti-TNP antibodies prevent effector cell lysis of TNP-modified target cells. With respect to the third mechanism, it is postulated that at least some of the H-2 region products retain their unaltered antigenic structure in that antibodies directed against the relevant unmodified K or D region products inhibit lysis of TNP-modified target cells by the effectors. The in vitro chemically modified autologous CML model provides evidence of major histocompatibility linked control of T-cell mediated immunity at two distinct functional levels. One level is expressed in the stimulating cell population and involves either the formation of new self-antigenic structures by modification of cell surface products of genes mapping in or near the serologically defined H-2 regions, or recognition of hapten specificities in close association with unaltered autologous H-2 products. The other level is expressed either in responding lymphocytes or in a population of accessory cells necessary for generating the CML and is defined as immune response potential to the modified or H-2-associated autologous specificities. It is controlled by Ir genes probably mapping in or near the I-A subregion.

19. CROSS-REACTIVE LYSIS OF TRINITROPHENYL (TNP)-DERIVATIZED H-2 INCOMPATIBLE TARGET CELLS BY CYTOLYTIC T LYMPHOCYTES GENERATED AGAINST SYNGENEIC TNP SPLEEN CELLS.

Burakoff SJ, Germain RN, Benacerraf B
Dept. Pathology, Harvard Medical Sch., Boston, MA 02115
J Exp Med; 144(6):1609-1620 1976

Cross-reactive lysis of trinitrophenyl (TNP) conjugated target cells was investigated using A/J (H-2a), B10.BR(H-2k), BALB/c(H-2d), C57BL/6B6(H-2b), and DBA/1(H-2q) mice. Normal spleen cells, when cultured with irradiated TNP-derivatized syngeneic spleen cells, developed cytotoxic effectors that lysed a TNP-derivatized target which was H-2 compatible with the effector. However, these effectors also lysed to a lesser extent, TNP tumor and TNP spleen targets that were H-2 incompatible. This cross-reactive lysis correlated with the degree of cytolysis seen on the TNP-derivatized syngeneic target; it appeared to be mediated by Thy 1.2-bearing cells and was inhibited by antisera to the K and/or D loci of the target cell and not by antisera to non-K or non-D surface antigens. This high positive correlation between lysis of H-2 identical and H-2 nonidentical target cells mediated by the same effector population indicates that cross-reactive lysis is not an independent, randomly occurring event. Nonradiolabeled TNP-derivatized lymphoid cells syngeneic to either the stimulator or the target were able to competitively inhibit cross-reactive lysis, while TNP chicken RBC were unable to specifically inhibit lysis. These data on cross-reactive lysis of TNP-conjugated targets are consistent with the altered-self hypothesis. (19 refs)

20. INHIBITION OF CELL-MEDIATED CYTOLYSIS OF TRINITROPHENYL-DERIVATIZED TARGET CELLS BY ALLOANTISERA DIRECTED TO THE PRODUCTS OF THE K AND D LOCI OF THE H-2 COMPLEX (MEETING ABSTRACT).

Burakoff SJ, Germain RN, Benacerraf B
Dept of Pathology, Harvard Medical School, Boston, Ma 02115
Fed Proc; 35(3):826 1976

The role of the products of the K and D loci of the H-2 complex as target antigens for cytotoxic T cells generated to modified syngeneic cells has been investigated. We have confirmed the findings that normal spleen cells, when cultured with irradiated Tnp-derivatized syngeneic spleen cells, develop cytotoxic effectors that lyse most effectively a target cell that is: a) H-2 compatible with the stimulator; and b) Tnp derivatized. The cytolysis of these derivatized target cells is inhibited by: a) alloantisera directed to the products of the K and/or the D loci: and b) antihapten antibody, i.e, anti-Dnp antiserum. Effector cells generated to Tnp-derivatized stimulators also lysed, but less effectively, Tnp targets not sharing H-sD or H-2K products with the stimulator or effector cells; and this cross-reactive lysis occurred both for tumor and spleen targets. This cross-reactive lysis was also inhibited by antisera to the K and/or D loci of the Tnp-tumor or Tnp-spleen target cell. These data support the hypothesis that cytotoxic T cells in this model system are generated to new antigenic determinants which are determined by the modification of H-2 gene products. (Author Abstract)

21. BIFUNCTIONAL HISTOCOMPATIBILITY-LINKED REGULATION OF CELL MEDIATED LYMPHOLYSIS TO MODIFIED AUTOLOGOUS CELL SURFACE COMPONENTS. (PP. 133-157)

Shearer GM, Schmitt-Verhulst AM, Rehn TG
The Role of Products of the Histocompatibility Gene Complex in Immune Responses. Katz DH, Benacerraf B, ed. New York, Academic Press Inc, 1976.

Recent developments in the chemically modified autologous cell mediated lympholysis (CML) in vitro models are reviewed with respect to their relationship to products and functions of the major histocompatibility gene complex. Murine thymus-derived lymphocytes can be sensitized in vitro to trinitrophenyl-(TNP-) modified or N-(3-nitro-4-hydroxy-5-iodophenylacetyl)-beta-ananylglycylglycyl -(N-) modified autologous lymphocytes. During 5 days of culture, cytotoxic effector cells are generated which specifically lyse only Cr51-labeled spleen target cells which are modified with the same agent and which share H-2K and/or H-2D alleles with the cells of the sensitizing phase. Experiments using F1 hybrid responding lymphocytes sensitized and assayed with TNP-modified parental cells indicate that the homology required for lympholysis is either between modified stimulating and modified target cells, or among responding, modified stimulating and modified target cells. Specificity is such that effector cells generated by sensitization with TNP-modified stimulating cells are incapable of lysing N-modified, H-2-matched targets, and vice versa. Antisera directed against serologically defined region products of unmodified target cells inhibit lysis of TNP-modified target cells, whereas anti-Ia antibodies have no detectable effect on lysis. Anti-TNP sera also block the lysis of TNP-modified targets irrespective of whether the effector cells are specific for TNP-modified autologous H-2 products or unmodified H-2 alloantigens. Such experiments indicate that the effector cells are directed against specificities associated with serologically-defined region products and that some TNP groups are sterically close to the H-2K and H-2D region antigens. Sensitization of B10.A responding lymphocytes with TNP-modified autologous spleen cells generates effectors which lyse TNP-modified K-end matched targets, but not TNP-modified D-end matched target cells. In contrast, B10.D2 effector cells generated by TNP-autologous sensitization lyse TNP-modified K-end and H-2D region matched targets equally well. Experiments using (B10.AxB10.D2)F1 hybrid responding lymphocytes sensitized with TNP-modified F1 , B10.A or B10.D2 stimulating cells and the response patterns observed in other C57BL/10 congenic and recombinant mouse strains indicate that cell-mediated lympholysis response potential to TNP-modified autologous components controlled by genes mapping in or near H-2D are controlled by Ir genes mapping in or near H-2K and/or or I-A. These findings demonstrate bifunctional genetic regulation of cellular immunity to modified autologous lymphocytes in which genes mapping in or near the serologically defined regions of the major histocompatibility complex control new antigenic specificities, and genes mapping in or near the I region regulate response potential to these new self-antigens. A similar type of bifunctional H-2-linked control of immunity may be relevant in autologous systems for surveillance against viral-associated neoplasia and for autoimmunity. (28 refs)

22. BIFUNCTIONAL MAJOR HISTOCOMPATIBILITY-LINKED GENETIC REGULATION OF CELL-MEDIATED LYMPHOLYSIS OF TRINITROPHENYL-MODIFIED AUTOLOGOUS LYMPHOCYTES.

Schmitt-Verhulst AM, Shearer GM
Natl. Cancer Inst., Bethesda, MD 20014
J Exp Med; 142(4):914-927 1975

To determine the relative contribution of immune response potential and immunogenicity to the generation of lympholysis, the responses generated by lymphocytes from high and low responders and those generated by lymphocytes from an F1 (between high and low responder parents) against modified-parental and F1 stimulating cells were compared. Murine thymus-derived lymphocytes were sensitized in vitro to trinitrophenyl (TNP)-modified autologous spleen cells. Cytotoxic effector cells were generated which were specific for TNP-modified target cells expressing the same H-2K and H-2D serological regions as the modified stimulator cells. Spleen cells from two C57BL/10 congenic strains of mice sharing common I-C, S, and D regions, but differing at K, I-A, and I-B regions, generated different levels of lytic responses to the shared modified H-2Dd products on sensitization with autologous TNP-modified cells. Lymphocytes from an F1 (between responder and nonresponder strain) generated a level of cytolysis toward the H-2Dd modified specificity which was of the same order of magnitude as that obtained with the high responder, irrespective of whether F1 or either parental strain of modified stimulator cell was used. The results of these studies suggest that the modification of H-2Dd products results in the formation of new antigenic determinants in both parental strains. However, the difference in responsiveness observed appeared due to a gene or genes mapping in the K, I-A, or I-B region which influenced the ability of the responding lymphocytes to react to these modified H-2Dd products. The results imply bifunctional H-2-linked regulation of a T-cell-mediated immunity in the response potential to and in the formation of new, modified self-antigens.

23. FUNCTIONAL ACTIVITY OF CYTOLYTIC T LYMPHOCYTES IN THE REJECTION OF TUMOR TARGET CELLS.

Brunner KT, Cerottini JC
Dept. Immunology, Swiss Inst. Experimental Cancer Res.-ISREC, Ch. des Boveresses, 1066 Epalinges-sur-Lausanne, Switzerland
Ann Immunol (Paris); 128C(1/2):473-483 1977

The mechanism of the cytolytic T-lymphocyte CTL destruction of grafts and tumors is reviewed. In addition, the characteristics of CTL memory cells, the functional activity of CTL induced in secondary mixed leukocyte cultures (MLC-2) against allogeneic tumor growth in mice, and the specificity of CTL formed in response to Moloney sarcoma virus (MSV)-induced tumors are reported. Preliminary results indicate that memory CTL appear in the spleen and peripheral blood of mice 7 days after the ip injection of allogeneic tumor cells and persist in high concentration for at least 3 mo. The functional activity in vivo of CTL induced in MLC-2 was demonstrated by the simultaneous inoculation of irradiated (650 rads) C57BL/6 (H-2b) mice with cells from MLC-2 stimulated by spleen cells of DBA/2 (H-2d) mice and allogeneic P-815 (H-2d) tumor cells. The iv transfer of CTL induced in the MLC-2 protected the irradiated mice effectively against growth of the sc-injected allogeneic tumor cells. In demonstrating the immune-response specificity of CTL against tumors induced by MSV, highly reactive CTL were obtained from spleen cell

MLC-2 provided by C57BL/6 and DBA/2 mice immune to the MSV tumors. In vitro, the CTL obtained from the two different MLC-2 lysed the syngeneic MSV-induced tumor cells preferentially. The results show a genetic restriction related to the histocompatibility complex upon induction of CTL cytotoxic activity. (39 Refs)

24. STIMULATION OF MOUSE MIGRATION INHIBITORY FACTOR (MIF) PRODUCTION FROM MSV-IMMUNE LYMPHOCYTES BY SOLUBLE TUMOR-ASSOCIATED ANTIGEN: REQUIREMENT FOR HISTOCOMPATIBLE MACROPHAGES.

Landolfo S, Herberman RB, Holden HT
Lab. Immunodiagnosis, NCI, Bldg. 8, Room. 118,
Bethesda, MD 20014
J Immunol; 118(4):1244-1248 1977

Spleen cells from C57BL/6 mice immunized with murine sarcoma virus (MSV) are capable of producing migration inhibition factor (MIF) in response to stimulation with a specific tumor-associated antigen prepared by solubilization with 3 M KCl. This response is T-cell dependent. Further investigations into the reaction revealed that spleen cells from mice immunized with MSV did not produce MIF in response to antigen stimulation when depleted of macrophages. The ability of the spleen cells to produce MIF was restored by the addition of nonimmune syngeneic macrophages, but not by allogeneic macrophages, from peritoneal exudate cells. The inability of allogeneic macrophages to provide this function was not due to their increased suppressor activity since, in mixing experiments, they did not interfere with the ability of immune spleen cells to produce MIF. Furthermore, they were not defective since they could supply this cooperative function to appropriate F1 mice. The results indicate that macrophages are essential for stimulation of MIF by soluble tumor antigens, and that genetic similarity is required for efficient interaction between macrophages and T lymphocytes. (23 Refs)

25. THE ROLE OF IA ANTIGENS IN MLC STIMULATION. (PP. 159-165)

Sachs DH, Schwartz RH, Fathman CG
The Role of Products of the Histocompatibility Gene
Complex in Immune Responses. Katz DH, Benacerraf B,
ed. New York, Academic Press Inc, 1976.

To determine whether Ia antigens and cell surface antigens that stimulate mixed lymphocyte culture (MLC) reactions are closely associated at the cell surface and at the genetic level, the possible blocking of MLC reaction stimulation by Ia antibodies was studied. The (B10.AxA)F1 anti-B10 antiserum with a demonstrably high cytotoxic titer of anti-Ia.8 antibody (tested on B10.D2 lymphoid cells) specifically inhibited stimulation of B10.A responder cells by B10.D2 stimulators. The suppressed level of stimulation was comparable to that of (B10xB10.A)F1 responders to B10.D2, in which case there was a genetic block of the Ia antigen shared between B10 and B10.D2. This same antiserum had no effect on B10.D2 responder cells in the reverse MLC reaction. In addition, it blocked (B10.AxB10.D2)F1 stimulator cells from stimulating B10.A responders, but not B10.D2 responders, indicating that binding of antibodies to the nonrelevant parental antigens on the F1 cells did not interfere with stimulation by antigens of the other parental strain. These results are all consistent with the hypothesis that Ia alloantigens are responsible for stimulation of the Ir region associated MLC reaction. (10 refs)

26. SPECIFICITY IN T CELL MEDIATED LYMPHOLYSIS: IDENTICAL GENETIC CONTROL OF THE PROLIFERATIVE AND EFFECTOR PHASES OF ALLOGENEIC AND XENOGENEIC REACTIONS.

Peck AB, Alter BJ, Lindahl KF
Biomedical Center, Uppsala Univ., Uppsala, Sweden
Transplant Rev; 29:189-221 1976

Arguments supporting the hypothesis that the same mechanisms which control the development of cytotoxic T effector cells in allogeneic reactions also control their development in xenogeneic reactions are summarized. In both allogeneic (mouse anti-mouse) and xenogeneic (human anti-mouse) reactions, the recognitive phase, measured by resulting lymphocyte proliferation in mixed lymphocyte culture (MLC), is under the genetic control of two major mouse antigen systems: the H-2 associated lymphocyte-defined (LD) determinants and the products of the Mls locus. The stimulatory H-2 LD determinants are coded by genes in the H-2K, H-2 I and H-2D regions. Under standard culture conditions the H-2 I LD determinants induce the strongest MLC activation of responding cells. Evidence for stimulation of human lymphocytes by products of the mouse Mls locus is circumstantial because, in xenogeneic reactions, it is impossible to isolate only Mls-locus differences. Available data suggest, however, that among the non-H-2 antigens it is the mouse Mls locus which induces proliferation of human lymphocytes. Following MLC activation, the in vitro generation of cytotoxic T lymphocytes (CTL) in allogeneic and xenogeneic reactions is largely dependent on the gene products of the H-2K and H-2D regions. There is a good correlation in allogeneic reactions between cell-mediated lympholysis (CML) and the H-2 private specificities. The products of the two strongest MLC activating antigen systems, the H-2 I regions and the Mls locus, either do not generate effector cells or do so only at a barely detectable level. There appears, therefore, to be specialization of function for the H-2 I LD determinants and the H-2K and H-2D SD molecules in the production of CTL. It is concluded that these results might specifically implicate the H-2 private specificity as the actual target antigen of CTLs; on the other hand, this specificity may merely serve as an excellent marker for the true target.

27. GENETIC AND CELLULAR ASPECTS OF XENOGENEIC MIXED LEUKOCYTE CULTURE REACTION.

Lindahl KF, Bach FH
Inst. Genetics, 5 Koln 41, Weyertal 121, W. Germany
J Exp Med; 144(2):305-318 1976

The antigens that stimulate xenogeneic lymphocytes to induce proliferative responses in the mixed lymphocyte culture (MLC) are studied. Human peripheral blood lymphocytes were sensitized against spleen cells from mice; the lymphocytes were then restimulated with spleen cells of a variety of mouse strains that shared non-H-2 antigens. The greatest thymidine uptake occurred when there was restimulation with the cells from an H-2 identical strain or a specific primary stimulator. The strain specific antigens in the C57BL/10 background or the species specific antigens accounted for less than one-sixth of the total stimulation. The control of antigens that stimulated allogeneic and xenogeneic MLC responses was the same. The MLC responses required adherent cells and T-cells; however, in the mouse-human MLC, both of the cell types had to come from the human. The results indicated that the vigor of alloaggression could be explained by secondary potentiating mechanisms.

28. MIXED LYMPHOCYTE REACTIVITY AND CELL-MEDIATED LYMPHOLYSIS TO D-END DIFFERENCES OF THE MURINE MAJOR HISTOCOMPATIBILITY COMPLEX. COMPARISON OF IN VITRO RESPONSES TO EXCLUSIVE D-END OR MORE EXTENSIVE MHC DIFFERENCES.

Hodes RJ, Schmitt-Verhulst AM, Hathcock KS, Shearer GM

Natl. Cancer Inst., Building 10, Rm. 4B17, Bethesda, MD 20014

Scand J Immunol; 5(4):369-370 1976

In vitro mixed lymphocyte culture (MLC) responses and the in vitro induction of cell-mediated lympholysis (CML) were studied in congenic mouse strain combinations in which the responding and stimulating strains differed either at the entire major histocompatibility complex (MHC) or only at the D end of the MHC. In contrast to previously reported studies, the relative strengths of stimulation by 'D end only' differences or by whole MHC differences were examined by stimulating identical responding populations with titrated numbers of stimulating cells that differed from the responder either at the D end only or over the entire MHC. Isolated D-end differences were sufficient to generate significant MLC and CML responses in each combination tested. Several 'D end only' differences (the responses of B10.A to B10.A(2R); of B10.A(2R) to B10.A; of B10.D2 to B10.HTG; and of B10.HTG to B10.D2) were severalfold less efficient in stimulating MLC and CML responses than were control stimulating cells differing over the whole MHC. In contrast, when the mutant D-end allele da was present on the stimulating cell (the responses of B10.D2 to B10.D2(M504) and of B10 to B10.D2(R106)), stimulation by an isolated D-end difference was comparable to stimulation by broader MHC differences.

29. SPECIFIC BINDING OF K- AND I-REGION PRODUCTS OF THE H-2 COMPLEX TO ACTIVATED THYMUS-DERIVED (T) CELLS BELONGING TO DIFFERENT SUBCLASSES.

Nagy Z, Elliott BE, Nabholz M

Medical Res. Inst., Hungarian Acad. Science, Budapest XIV, Hungary

J Exp Med; 144(6):1545-1553 1976

The specific binding of allogenic K- and I-region products by responder T blasts was studied under conditions where these antigens are presented together on separate stimulator cells. Responder cells, (C57BL/6J x A.TL)F1 lymph node cells depleted of B cells by filtration through nylon wool columns, were activated against incompatible K-region and I-region products. The resulting T blasts were stained with different concentrations of antisera directed against incompatible stimulator K-region or I-region products, or both. The results suggest that in these cultures, each activated responder blast stains with antiserum directed against either K-region or I-region products, but not both. Responder blasts from the same cultures were treated with antiserum and complement (C) directed against either Ly-1.2 or Ly-2.2 T cell-specific surface antigens. Anti-Ly-1.2 serum and C specifically eliminated virtually all responder blasts staining with antiserum directed against stimulator I-region products. Anti-Ly-2.2 serum reduced the proportion of cells staining with antiserum against stimulator K-region products to background levels. These results suggest that T cells binding either stimulator K-region or I-region products belong to two different subclasses distinguishable by their Ly phenotypes. Possible explanations for this association of T cell subclass and specificity are discussed. (19 refs)

II. CELLULAR AND MOLECULAR BASIS OF IMMUNE RESPONSE GENE CONTROL

A. Genetic Basis of Immune Reactivity to Non-Tumor Antigens in Animal Systems

30. GENETIC CONTROL OF IMMUNE RESPONSES IN VITRO. III. TOLEROGENIC PROPERTIES OF THE TERPOLYMER L-GLUTAMIC ACID^{60}-L-ALANINE^{30}-L-TYROSINE^{10} (GAT) FOR SPLEEN CELLS FROM NONRESPONDER (H-2**S AND H-2**G) MICE.

Kapp JA, Pierce CW, Benacerraf B

Dept. Pathology, Harvard Medical Sch., Boston, MA, 02115

J Exp Med; 140:172-184 1974

The effect of the synthetic random terpolymer L-glutamic acid^{60}, L-alanine^{30}, L-tyrosine^{10} (GAT) on the GAT-specific immune response to GAT complexed to methylated bovine serum albumin (MBSA) in vivo and the effect of soluble GAT on the immune response to GAT-MBSA by spleen cells in vitro were investigated. Although nonresponder, H-2**s and H-2**g, mice failed to develop GAT-specific plaque-forming cell (PFC) responses to GAT, they did develop GAT-specific PFC responses when stimulated by GAT complexed to an immunogenic carrier such as MBSA. Injection of nonresponder mice with GAT specifically decreased their ability to develop anti-GAT PFC responses to a subsequent challenge with GAT-MBSA. Addition of GAT to cultures of spleen cells from nonresponder mice also prevented development of the GAT-specific PFC responses stimulated by GAT-MBSA. It is concluded that the interaction of nonresponder spleen cells with GAT leads to the induction of unresponsiveness in vivo and in vitro. (16 Refs)

31. GENETIC CONTROL OF THE MURINE CELL-MEDIATED IMMUNE RESPONSE IN VIVO. I. H-2-LINKED RESPONSIVENESS TO THE TERPOLYMER L-GLUTAMIC ACID^{60}-L-ALANINE^{30}-L-TYROSINE^{10} .

Davis S

Tufts-New England Medical Center, 171 Harrison Ave., Boston, Mass.

Scand J Immunol; 4(3):253-257 1975

Various inbred and congenic strains of mice were immunized sc with 50 micrograms of the linear terpolymer L-glutamic acid^{60}-L-alanine^{30}-L-tyrosine^{10} (GAT). Using a radioisotopic footpad assay to measure cell mediated immunity in vivo, the mice with H-2a, H-2b, H-2d, and H-2k histocompatibility alleles showed a positive immune response, whereas mice with H-2p and H-2s alleles failed to respond. The ability to elicit a positive local adoptive transfer reaction was found to reside in the T lymphocyte, B cells failing to give a local adoptive transfer response. Responder mouse T cells differed in their degree of immune responsiveness, BALB/c (H-2d) and B10.BR (H-2k) mice consistently giving the greatest reactivity. Unfractionated spleen cells from H-2q DBA-1 nonresponder mice showed no reactivity, although nylon wool-column-purified splenic T cells from DBA-1 mice yielded small but consistent adoptive immune response; this activity could not be demonstrated with purified T cells from other nonresponder strains. The data indicate that strain differences exist in the degree of cellular immune responsiveness. It is possible that nonresponder strains of mice differ in degrees and/or mechanism of unresponsiveness and that the immune response to GAT may well be quantitative rather than qualitative.

32. MULTIPLE H-2-LINKED IMMUNE RESPONSE GENE CONTROL OF H-2D-ASSOCIATED T-CELL-MEDIATED LYMPHOLYSIS TO TRINITROPHENYL-MODIFIED AUTOLOGOUS CELLS: IR-LIKE GENES MAPPING TO THE LEFT OF I-A AND WITHIN THE I REGION.

Schmitt-Verhulst AM, Shearer GM
Immunology Branch, Natl. Cancer Inst., Bethesda, MD 20014
J Exp Med; 144(6):1701-1706 1976

The Ir murine gene controlling cell-mediated lympholysis (CML) to trinitrophenyl (TNP)modified H-2Dd was mapped in the murine H-2 major histocompatibility complex (MHC) by using recombinant mouse strains on the A and B10 backgrounds. The mice were B10.A, B10.BR, SJL/J, A.TH, A.TL, A.AL, B10.S, and B10.HTT. Regardless of the genetic background, the s and k haplotypes at the K end generated high and low cytotoxic responses, respectively, to H-2Dd-TNP. The intermediate responder and low responder status of the A.TL and A.AL strains, respectively, indicated that a gene mapping in the K region of H-2 influences response potential. The differences in the levels of cytotoxicity in the A.TH and A.TL strains suggested an additional I region influence. These data suggest that multiple genes mapping within different regions of the MHC control the level of T cell-mediated cytotoxicity to chemically modified autologous cells. (22 refs)

33. UNIGENIC AND MULTIGENIC I REGION CONTROL OF THE IMMUNE RESPONSES OF MICE TO THE GAT^{10} AND GLF-GLT TERPOLYMERS.

Merryman CF, Maurer PH, Stimpfling JH
Thomas Jefferson Univ., Philadelphia, Pa. 19107
Immunogenetics; 2(5):441-448 1975

Recombinant strains of mice with known alleles in the I region of the H-2 complex were used to map the H-2 linked immune response genes controlling responsiveness to random terpolymers GAT^{10} and GLPhi. The Ir-GAT gene was mapped to either the IA or IB subregions. In contrast, data obtained in the GLPhi-GLT system indicated multigenic control. The responsiveness of the B10.A(3R), B10.A(5R), and B10.S(9R) recombinants indicated that 1 immune response gene, Ir-GLPhi-GLT.4 , mapped to the right of IB, i.e, in the IC subregion. The nonresponsiveness of the B10.A(1R), B10.A(2R), B10.M(17R), and AQR mice having responder ICd alleles but IAk-IBk nonresponder alleles and the positive response of a (C57BL/6 x A/J)F1 hybrid derived from 2 nonresponder parental strains indicated the presence of a second gene in IA-IB subregions, Ir-GLphi-GLTB. The interaction between these 2 genes, each present in a different I subregion, controls the immune response.

34. COMPLEMENTATION OF H-2-LINKED IR GENES IN THE MOUSE.

Dorf ME, Benacerraf B
Dept. Pathology, Harvard Medical Sch., Boston, MA, 02115
Proc Natl Acad Sci USA; 72(9):3671-3675 1975

The immune response to the random linear terpolymer of L-glutamic acid, L-lysine, and L-phenylalanine (GLPh) is under dominant histocompatibility-2 (H-2)-linked immune response (Ir) gene control in the mouse. Matings between two nonresponder strains produced responder F1 hybrids, demonstrating complementation of the nonresponder alleles. This observation, coupled with the fact that several intra H-2 recombinant strains derived by recombination between two nonresponder parental haplotypes are also GLPh responders, indicated at least two dominant loci are concerned with responsiveness to this terpolymer. The complementary genes were termed alpha (tentatively localized in a new subregion of the H-2 complex, I-F) and beta, which maps in the I-A subregion. Generally, both the alpha(+) and the beta(+) alleles are required for responsiveness. However, in the (C57BL/6J x SJL)F1 hybrid, complementation occurred between two parental nonresponder strains, each of which carried beta genes derived from different H-2 haplotypes, yet lacked functional alpha genes not noted. The possible cell levels at which these genes may function in the regulation of the immune response are discussed. (Author abstract) (22 Refs)

35. REQUIREMENT FOR TWO H-2 COMPLEX IR GENES FOR THE IMMUNE RESPONSE TO THE L-GLU, L-LYS, L-PHE TERPOLYMER.

Dorf ME, Stimpfling JH, Benacerraf B
Dept. Pathology, Harvard Medical Sch., Boston, MA, 02115
J Exp Med; 141:1459-1463 1975

In a previous report, using histocompatibility-2 (H-2) recombinant mice, the immune response gene (Ir) for the linear synthetic terpolymer of L-glutamic acid, L-lysine, and L-phenylalanine (GLPh) was mapped within the I region of the H-2 complex, specifically in the region termed I-B. The requirement for at least two H-2-linked Ir-GLPh genes for GLPh responsiveness is reported. All inbred mice and F1 hybrids were between 8 and 30 wk of age at the beginning of immunization. The mice were bled 7 days after secondary immunization. That the mating of two nonresponder strains produced responder F1 hybrids was viewed as evidence of complementation of the nonresponder H-2**a and H-2**b alleles. Observation of recombinant strain responsiveness indicated that one of the Ir-GLPh genes could tentatively be localized in the chromosomal region between I-B and D. The other Ir-GLPh gene was thought to lie to the left of the I-C region. Each of the genes is thought to be dominant, but both alleles must be present for expression of GLPh responsiveness. (18 Refs)

36. GENETIC CONTROL OF THE IMMUNE RESPONSE: A SELECTIVE DEFECT IN IMMUNOLOGIC (IGG) MEMORY IN NONRESPONDER MICE.

Grumet FC
Stanford U. Sch. Med., California
J Exp Med; 135(1):110-125 1972

The kinetics of antibody formation after immunization with the synthetic polypeptide poly-L(Tyr,Glu)-poly-D,L-Ala - poly-L-Lys ¢(T,G)-A - L in aqueous solution were studied in genetically high responder (H-2b) and non-responder (H-2k) inbred mice of strain C3H.SW. Both strains showed equally good primary responses consisting of rapid formation of IgM antibody during the first wk postimmunization. However, after booster injections on day seven (secondary antigen challenge) and day 30 (tertiary antigen challenge) post primary immunization, striking differences between responder and non-responder mice were seen. Responders rapidly developed high titers of IgG antibody, while non-responders produced almost none. Non-responder mice also failed to produce a significant increase in their IgM antibody after secondary and tertiary antigen challenge. Characterization of the antibody response on the basis of column chromatography and reactivity with specific antisera indicated that the antibodies belong to the IgM class of immunoglobulins. The data are consistent with the hypothesis that the immune response gene effect is exerted through thymus-derived cells which influence the conversion of antibody-producing, bone-marrow-derived B-cells from IgM to IgG production.

37. ASSOCIATION OF H-2 TYPES WITH GENETIC CONTROL OF IMMUNE RESPONSIVENESS TO IGA ALLOTYPES IN THE MOUSE (MEETING ABSTRACT).

Lieberman R, Humphrey W

Natl. Inst. Allergy Infect. Dis., Natl. Inst. Hlth., Bethesda, Md.

Proc Natl Acad Sci USA; 68(10):2510-2513 1971

The immune response to BALB/c IgA myeloma proteins (Ir-IgA) in inbred strains of mice of various H-2 types was studied. Mice from five different linkage groups were immunized with IgA myeloma proteins, which carry allotypic determinants A on their heavy-chain constant region (CH) and myeloma specific or idiotypic determinants on their Fab region. Four sc protein injections distributed over six sites were given. Following immunization, the mice were bled twice weekly to obtain antisera. Antibody assays included double diffusion agar gel plate analyses and passive hemagglutination studies. The immune response of the inbred mice ranged from good to poor to none, depending on the H-2 type of the recipient strain. H-2 types a, k, s, and r responded well while H-2 types b, d, v, l, and q responded poorly or not at all. Since the immune response to IgA myeloma proteins was found to be associated with specific H-2 types, appropriate congenic strains were then similarly immunized to prove association of the Ir-IgA to the H-2 locus. Six congenic strains of mice from a2 and a4 immunoglobulin heavy-chain linkage groups, comprising four different H-2 types (a, k, d, and b), were immunized with BALB/c IgA myeloma protein. Corroboration of the preceeding results was obtained. Finally, two recombinant strains of mice were immunized with the myeloma protein. Resultant chromosome mapping of the genes controlling the Ir-IgA showed that it is essentially in the same region as the Ir-1 of McDevitt and appeared to be on the right side of Ss, which itself is on the right side of the autosomal chromosome. However, a more precise localization is pending. It should be noted that most antibodies, especially in the congenic and recombinant strains of mice, were directed to Fab idiotypic specificities. This suggests that antibody response to CH determinants is controlled by genetic systems other than or including H-2.

38. H-2-LINKED IR GENES CONTROLLING IMMUNE RESPONSES TO BALB/C IGG (GAMMA 2A) MYELOMA PROTEINS HAVING KAPPA OR LAMBDA LIGHT CHAINS.

Lieberman R, Paul WE, Humphrey W, Potter M

Natl. Cancer Inst., Bethesda, Md.

Transplant Proc; 7(1/Suppl. 1):115-118 1975

The possibility that the same H-2 linked Ir gene is involved in immune responses to all BALB/c immunoglobulin G (IgG) proteins of the same class was investigated. Inbred, H-2-cogenic, and recombinant strains were studied for their response to four kappa-type IgG myeloma proteins (MOPC173, LPC1, UPC10, and RPC5) and two lambda-type myeloma proteins (HOPCO and S5444). No antibody response was observed in inbred mice with the same allotype as BALB/c. In strains with allotypes different from BALB/c, a high immune response was found in H-2 types b, r, s, and v for two to three of the kappa-type myeloma proteins and for both the lambda types. Results with B10-cogenic strains and H2a/H2b recombinant strains indicated that regardless of the VH region of the myeloma protein, the same Ir gene (Ir-1b) is employed in the immune response to BALB/c kappa-type myeloma proteins. A high response to the kappa myeloma proteins was found in H-2b (B10) and a low response in H-22 (B10.A). Among the H-2a/H-2b recombinants, B10.A (1R) and B10.2 (2R) gave a low response and B10.A (4R) and B10.A (5R) gave a high response. The response of 4R for UPC10 or RPC5 was less than that of 5R or B10. All the recombinants tested gave little or no response to the lambda-type protein S5444, while B10 gave an excellent response. That the immune response to HOPC1 was linked to H-2 was shown by the very high response of B10.M mice to this protein. The recombinants also gave low responses to HOPC1. The data support the concept that genetic control of responsive to kappa-type IgG myeloma proteins involves recognition of constant-region allotypic-associated determinants.

39. GENETIC STUDIES IN INBRED RATS. VI. LINKAGE RELATIONSHIPS OF MIXED LYMPHOCYTE REACTIVITY, SEROLOGICALLY DEFINED ANTIGENS (AG-B, AG-C) AND THE IMMUNE RESPONSE TO POLY(GLU^{52} LYS^{33} TYR^{15}).

Shonnard JW, Cramer DV, Poloskey PE, Davis BK, Gill TJ

Univ. Pittsburgh Sch. Medicine, Pittsburgh, PA 15261

J Immunogenet; 3(1):61-70 1976

In order to investigate the structure of the rat major histocompatibility complex, the Ag-B allotype, mixed lymphocyte reactivity (MLR) and the immune response to poly(Glu^{52} Lys^{33} Tyr^{15}) were assayed in male rats from the F2 hybrid and two backcross generations of the F344 and DA strains. No disparity between Ag-B type and mixed lymphocyte reactivity was found in 263 animals. The immune response to poly(Glu^{52} Lys^{33} Tyr^{15}) was closely linked to the Ag-B locus. Both antibody production and the delayed hypersensitivity response were under polygenic control.

40. HISTOCOMPATIBILITY-LINKED GENETIC CONTROL OF DISEASE SUSCEPTIBILITY. MURINE LYMPHOCYTIC CHORIOMENINGITIS VIRUS INFECTION.

Oldstone MB, Dixon FJ, Mitchell GF, McDevitt HO

Scripps Clin. Res. Fdn., La Jolla, Calif.

J Exp Med; 137(5):1201-1212 1973

The susceptibility of murine strains to acute lymphocytic choriomeningitis (LCM) virus infection was genetically determined and the question of whether the tendency for increased susceptibility was linked to the major histocompatibility (H-2) locus was studied. Acute necrotizing inflammatory disease after intracerebral injection of LCM virus is largely dependent on the host immune response to the virus and is controlled partly by a dominant gene which is closely linked to the H-2 locus. The F1 hybrid (H-2q/k) from mating a susceptible SWR/J mouse (H-2q/q) to a resistant C3H/HeJ mouse (H-2k/k) is susceptible to LCM virus disease. When such hybrids (H-2q/k) are backcrossed to susceptible parents (H2-q/q), all F2 offspring (H-2q/q, H-2q/k) backcross to resistant parents (H2-k/k) results in half of the F2 offspring being susceptible (H-2q/k) while the other half are resistant (H-2k/k). Similarly, in congenic H-2q/q and H-2k/k mice, H-2q/q mice are relatively susceptible to acute LCM disease, whereas H-2k/k are resistant.

41. DISSECTION OF THE GENES OF THE MAJOR HISTOCOMPATIBILITY COMPLEX.

Bach FH, Schendel DJ, Widmer MB, Alter BJ, Bach ML

Dept. Medical Genetics, Univ. Wisconsin, Madison, WI 53706

Adv Biosci; 12:63-69 1974

Certain genetic and cellular aspects of two study systems are discussed: mixed leukocyte culture (MLC) as an in vitro model of the recognitive phase of the homograft reaction; and the cell-mediated lympholysis (CML) test to represent the destructive phase. In the mouse, the genes which lead to the proliferative phase in mixed culture are not the same as the serologically defined (SD) antigens. Furthermore, although lymphocyte-defined (LD) differences activate the MLC, the cytotoxic lymphocytes (CLs) do not in all cases use these LD differences as a target. In a mixed culture between mouse strains AQR and B10.T(6R) (which differ only by LD components of H-2), SD appeared to be the target event although LD activated the mixed culture. A model is proposed wherein both LD "antigens" and SD antigens are present on a target lymphocyte. When cells differ by both LD and SD, the responding cells include one population of presumed T lymphocytes which respond to LD by proliferating. These cells are referred to as proliferating helper cells (PHCs). Given PHC activation, a second population of cells can be activated against the SD antigens and show cytotoxicity against the target cells because of the SD antigens. Preliminary in vitro data are consistent with the existence of two such separate cell populations responding to LD and SD.

42. MAJOR HISTOCOMPATIBILITY-LINKED GENETIC CONTROL OF MURINE CELL-MEDIATED CYTOTOXICITY TO TRINITROPHENYL-MODIFIED SYNGENEIC LYMPHOCYTES.

Shearer GM
Natl. Cancer Inst., Bethesda, Md. 20014
Transplant Proc; 7(1,Suppl1):109-113 1975

A study was undertaken to demonstrate effector-cell specificity to trinitrophenyl (TNP)-modified H-2K or H-2D serological regions and probable H-2-linked genetic control of responsiveness to a specificity resulting from TNP modification of the H-2D serological region of B10.A spleen cells. The results of this study indicate that TNP modifies cell surfaces, presumably proteins, that are controlled by genes within the H-2K and/or H-2D serological regions of the murine major histocompatibility complex (MHC). These proteins are conformationally changed, creating new antigenic determinants that serve both as stimulating immunogens and target antigens. H-2-linked Ir genes expressed by T cells appeared to control response potential to the new antigenic determinants created by these TNP-induced alterations on the gene products of H-2K and H-2D. It is suggested that the H-2K and H-2D regions of the MHC control the synthesis of cell-surface glycoproteins which function to serve in the syngeneic model as easily modifiable autologous surface components. It appears that not only are many Ir-controlled responses H-2-linked, but that H-2 plays a significant part in the response potential to and in formation of new antigenic determinants.

43. CELL-MEDIATED LYMPHOLYSIS OF N-(NITRO-4-HYDROXY-5-IODOPHENYLACETYL)-B-ALANYLGLYCYLGLYCYL-MODIFIED AUTOLOGOUS LYMPHOCYTES: EFFECTOR CELL SPECIFICITY TO MODIFIED CELL SURFACE COMPONENTS CONTROLLED BY THE H-2K AND H-2D SEROLOGICAL REGIONS OF THE MURINE MAJOR HISTOCOMPATIBILITY COMPLEX.

Rehn TG, Shearer GM, Koren HS, Inman JK
Immunology Branch, Natl. Cancer Inst., Bethesda, MD 20014
J Exp Med; 143(1):127-142 1976

N-(3-nitro-4-hydroxy-5-iodophenylacetyl)-b-alanylglycylgylcyl (hereafter referred to as N)-modified autologous lymphocytes were used for the in vitro sensitization of splenic lymphocytes from four C57BL/10 congenic mouse strains. N-modified phytohemagglutinin-stimulated spleen cells or N-modified tumor cells were used to assay the effector cells generated after 5 d of culture. In order for lysis to occur, both N-modification of the targets and H-2 homology were required. Effector cells lysed N-modified target cells only when there was homology at either or both ends of the major histocompatibility complex (MHC) between stimulator and target cells. B10.BR effector cells lysed targets sharing alleles at K (or K + I-A) and/or at D. B10.A effector cell specificity was mapped to K (or K + I-A) and/or the D half of the MHC (D or D + I-C and/or S). For B10.D2 cells the specific regions were determined as D (or D + S + I-C) and a region not including D of the MHC. C57BL/10 effector cells lysed N-modified targets only when there was target cell H-2 homology at K, I-A, and I-B or at the D serological region. As in the case of trinitrophenyl- (TNP) modified cells, B10.BR and B10 effector cells lysed targets sharing K and H-2 serological regions greater than target cells sharing D-end serological regions. C57BL-10 effector cells reacted greater to the K end than to the D end, as opposed to the equal reactivity for effector cells of this strain in the TNP system. The results fit the hypothesis that the antigen recognized by the effector cell includes an altered H-2 serological cell surface component. That the reaction is not hapten-specific and the H-2 homology is required only for effector:target cell interaction was excluded by use of two F1 combinations in which lysis of only N-modified target cells sharing the H-2 haplotype with the stimulating parental strain was obtained. N and TNP modification create distinct new antigenic determinants, since an effector cell sensitized to only one modifying agent will lyse only H-2 matched target cells modified with the same modifying agent. (17 refs)

44. T-LYMPHOCYTE-ENRICHED MURINE PERITONEAL EXUDATE CELLS. II. GENETIC CONTROL OF ANTIGEN-INDUCED T-LYMPHOCYTE PROLIFERATION.

Schwartz RH, Paul WE
Lab. Immunology, Natl. Inst. Allergy and Infectious Diseases, NIH, Bethesda, MD, 20014
J Exp Med; 143:529-540 1976

Antibody responses of peritoneal exudate cells from various mouse strains that had been enriched for T lymphocytes by passage over nylon wool columns (PETLES) were determined for three groups of antibodies: two linear random terpolymers, two branched chain copolymers, and the protein antigens porcine lactate dehydrogenase H4, staphylococcal nuclease, and the IgA myeloma protein TEPC 15. Response of these cells, with the exception of one case, was the same as that determined in vivo, thus indicating that the assay is an accurate measure of T lymphocyte function. Distribution patterns of responsiveness showed an association between immune response (Ir) genes and the major histocompatibility complex (MHC). Using recombinant strains heterozygous for MHC haplotypes, the Ir genes controlling the T lymphocyte proliferative responses to one of the branched chain copolymers and to TEPC 15 were localized to the K or I-A region of the MHC. It appears that the same Ir gene controls the expression of both the proliferative and antibody responses. The relationship between Ir genes and B cells is discussed. (24 Refs)

45. HISTOCOMPATIBILITY-LINKED IMMUNE RESPONSE GENE FUNCTION IN GUINEA PIGS. SPECIFIC INHIBITION OF ANTIGEN-INDUCED LYMPHOCYTE PROLIFERATION BY ALLOANTISERA.

Shevach EM, Paul WE, Green I
Lab. Immunology, Natl. Inst. Allergy and Infectious Disease, NIH, Bethesda, MD, 20014
J Exp Med; 136:1207-1221 1972

Immune response, as measured by T lymphocyte proliferation, to the antigens dinitrophenyl (DNP) conjugated to a copolymer of L-glutamic acid (60%) and L-lysine (40%) (GL) in strain 2 guinea pigs, and to a copolymer of L-glutamic acid (50%) and L-tyrosine (50%) (GT) in strain 13, is under control of immune response (Ir) genes. Strain 2 guinea pigs lack the strain 13 Ir gene; strain 13 guinea pigs lack the strain 2 Ir gene. Immune response of these animals was determined by immunizing strain 2 with 25 microg DNP-GL and strain 13 with 125 microg GT in each foot pad. Alloantisera prepared against lymph node and spleen cells of the other strain inhibited response of peritoneal exudate lymphocytes (PELS), which are primarily T lymphocytes, from each strain to the appropriate antigen, but to a lesser extent response to the antigens, purified protein derivative of tuberculin or phytohemagglutinin. When PELS were incubated with antigen (antigen-pulse) and alloantisera, no inhibition of lymphocyte response was seen. PELS from (2 x 13) F1 animals were inhibited in their response to each antigen by the appropriate alloantiserum, were inhibited to both antigens by the appropriate alloantiserum, and were inhibited to both antigens when treated with both alloantisera. Absorption of alloantisera with gamma globulin ruled out the possibility that the inhibition could have been secondary to anti-immunoglobulin allotype antibodies. Ir genes produce a cell-surface-associated product which is involved in antigen recognition by T lymphocytes. Mechanisms by which alloantisera might block this recognition are discussed. (42 Refs)

46. GENETIC CONTROL OF SPECIFIC IMMUNE RESPONSES.

Benacerraf B
Dept. Pathology, Harvard Medical Sch., Boston, MA 02115
Ann Immunol (Paris); 128C(1/2):385-392 1977

The genetic complex which controls the rejection of major histocompatibility locus (MHC)-HLA allografts in humans, and H-2 allografts in mice, also controls other immune defense mechanisms. The genes which control these two types of specific responses are found in region I of the MHC. This region is equally responsible for the graft vs host reaction, production of T cell antigen by macrophages, and contribution of Ia antigens to macrophages in the specific stimulation of T lymphocytes. Genes Ir and Is have their loci in regions I-A and I-C. Control of immune response by genes Ir was demonstrated for a number of antigens (terpolymers of glutamic acid, lysine and phenylalanine). Genetic control by Is genes was shown for the copolymer of glutamic acid and tyrosine. (29 Refs)

47. OVERVIEW OF RECENT ADVANCES IN FUNDAMENTAL IMMUNOLOGY. (P.59-74)

Benacerraf B, Katz DH
Immunological Aspects of Neoplasia: A Collection of Papers Presented at the Twenty-Sixth Annual Symposium on Fundamental Cancer Research, 1973. Baltimore,Williams and Wilkins Company, 1975.

Important recent advances in the field of cellular immunology are summarized. A major discovery was the recognition of two pathways for the differentiation of antigen-reactive cells. One of the largest problems in immunobiology is the understanding of the precise mechanism of T cell-B cell interaction in humoral responses. The Hapten-Carrier model is discussed. The mechanism of the allogeneic effect, which results from the injection of an appropriate number of allogeneic immunocompetent T cells to animals primed to hapten-protein conjugates, emphasizes the necessity for close T cell-B cell interaction when B cell binds antigen for an appropriate response to result. The biological significance of the regulatory influence of T cells on antibody production and the mechanisms of T cell regulation on the stimulation of B cells by antigen are considered. Recent experiments have demonstrated an identical gene(s) is probably expressed in both cooperating T and B cells in hapten-carrier response. This gene(s) is possibly concerned with the major histocompatibility H-2 region. The lack of cooperation between allogeneic A/J and BALB/c cells which share identical D region specificities demonstrates that the existence of common histocompatibility specificities at the D end will not permit T cell-B cell cooperation so that the K end of H-2 is presumably of crucial importance in this phenomenon. The demonstration of effective cooperation between semisyngeneic T and B cells shows that the presence of distinct major locus histocompatibility specificities in the two cells does not interfere with cooperation. Mechanisms of specific immunological tolerance are considered.

48. IMMUNE RESPONSE GENES IN RELATION TO DISEASE. (PP. 255-262)

McDevitt HO
The Role of Immunological Factors in Infectious, Allergic and Autoimmune Processes Miles International Symposium Series Number 8. Beers RF, Basset E, ed. New York, Raven Press, 1976.

The natures of several associations between histocompatibility determinants and susceptibility or resistance to specific diseases in animal models is discussed. In addition to an association between the murine major transplantation (H-2) antigen complex and resistance to virus-induced leukemogenesis, a definite association has been demonstrated between H-2 type and susceptibility to murine autoimmune thyroiditis, and the acute lymphocytic choriomeningitis virus infection. An association was also noted between the rat major histocompatibility complex (H-1) and susceptibility to experimental allergic encephalomyelitis. A very large number of associations between human histocompatibility determinants and a variety of autoimmune, neoplastic, and possible infectious diseases as well as diseases of unknown etiology has also been described. There is strong evidence in the cases of autoimmune thyroiditis in the mouse and ragweed hay fever in man which supports the conclusion that these associations are due to the effect of immune response genes. Preliminary genetic and epidemiological evidence suggests that many associations between human leukocyte antigens and disease are due to the effect of specific immune response genes which are perhaps important in influencing susceptibility or resistance to a wide variety of neoplastic, autoimmune, and infectious diseases.

49. THE HISTOCOMPATIBILITY-LINKED IMMUNE RESPONSE GENES.

Benacerraf B, Katz DH
Dept. Pathology, Harvard Medical Sch., Boston, MA, 02115
Adv Cancer Res; 22:121-173 1975

The literature on histocompatibility-linked immune response (Ir) genes is reviewed. Chapter headings include: immune responses controlled by histocompatibility-linked Ir genes; the mapping of mouse H-linked Ir genes; relationship of H-linked Ir genes to immunoglobulin structural genes; the cell type where histocompatibility-linked Ir genes are expressed; the demonstration of suppressor T cells specific for GAT^{10} in genetic nonresponder mice; the role of histocompatibility gene products in T-B cell interactions; identification of I region gene products; relationship of histocompatibility gene products to each other in nature and function; function of products of H-linked Ir genes and other I region gene products; relationship of these products to activation of immunocompetent cells; and the histocompatibility-linked Ir genes and disease. Each topic discussed is well-documented; a wide list of references is provided. (101 Refs)

B. Studies of Lymphocyte Subclasses Regulating Immunity to Antigens in Animal Systems

50. GENETIC CONTROL OF THE IMMUNE RESPONSE. THE EFFECT OF THYMECTOMY ON THE PRIMARY AND SECONDARY ANTIBODY RESPONSE OF MICE TO POLY-L (TYR,GLU)-POLY-D, L-ALA - POLY-L-LYS.

Mitchell GF, Grumet GC, McDevitt HO
Stanford U. Sch. Med., Stanford, Calif.
J Exp Med; 135(1):126-134 1972

The effect of thymectomy on the genetically controlled murine immune response-1 (Ir-1) to the synthetic polypeptide poly-L(Tyr,Glu)-poly-D,L-Ala-poly-L-lys (T,G)-A - L was studied with both aqueous and adjuvant immunization regimens. Adult thymectomy (combined with irradiation and bone marrow transfusion did not affect the aqueous antigen-induced (IgM) primary response of either high or low responder mice, but did abate the (IgG) secondary or tertiary response, a response which is restricted to the high responder strains. Adult thymectomy also blocked the normal high response to (T,G)-A - L in Freund's adjuvant in high responder mice and the high response to methylated bovine serum albumin (MBSA)-(T,G)-A - L in low responder mice. Neonatal thymectomy was also effective in blocking the response to (T,G)-A - L in Freund's adjuvant in high responder mice. These data are consistent with the concept that the Ir-1 gene effect is mediated via thymus cell interaction with antigen and with "B"-cells during the time of induction of IgG antibody formation.

51. OVERCOMING IR GENE CONTROL OF THE RESPONSE TO THE LYSOZYMES. (PP. 537-543)

Hill SW, Yowell RL, Kipp DE, Scibienski RJ, Miller A, Sercarz EE
Immune Reactivity of Lymphocytes Development, Expression, and Control. Feldman M, Globerson A, ed. New York, Plenum Press, Advances in Experimental Medicine and Biology, vol. 6, 1976.

The overcoming of Ir gene control of the response to lysozymes is described. A significant number of B-clonotypes was available for triggering throughout the H-26 mouse, furthermore, through local footpad immunization, the clonotypes were induced to make both immunoglobulin G and M antibody in the popliteal node; the memory cells that migrated to the spleen were stimulated to antibody formation by soluble antigen. In the spleen, the regulation in H-26 strains did not allow stimulation by lysozymes to advance further than the beginning suppressive phase. It is concluded that the response is able to progress to a cooperative, helper phase, because of access to antigen prior to the spleen and a different mixture of cells in the popliteal lymph node. (7 refs)

52. CELLULAR RESPONSES TO MURINE ALLOANTIGENS OF THE MAJOR HISTOCOMPATIBILITY COMPLEX. THE ROLE OF CELL SUBPOPULATIONS THAT EXPRESS DIFFERENT QUANTITIES OF H-2-ASSOCIATED ANTIGENIC MARKERS.

Plate JM
Massachusetts General Hosp., Boston, MA 02114
Eur J Immunol; 6(3):180-187 1976

An anti-H-2 serum produced between H-2 congenic mouse lines that differ with respect to their H-2K, Ir-1A and Ir-1B regions was used to study the functions of Ia-bearing cells in mixed lymphoid cell reactions and in the generation of cytotoxic killer T (Tk) cells. The strain selected as the target of this antiserum also differed from the immunizing donor with respect to its H-2K region but shared at least one common Ia antigen. Two procedures were used to study the effect of anti-Ia serum on lymphoid cell functions: a cell pellet of 1.5 x 10^7 or 2.0 x 10^7 lymph node cells was resuspended in 1.0 ml of antiserum at a dilution of 1:5 and incubated for 15 min, after which 1.0 ml of diluted rabbit serum that had been absorbed with mouse tissue was added and the mixture incubated for 45 min; and cells activated in vitro with mitogens or allogeneic lymphocytes were distributed in 12 x 75 mm tubes, specified dilutions of antisera were added, and the cells incubated for 15 min at either 0 C or 37 C. An equal volume of diluted rabbit serum was added, and the mixture incubated for 45 min. The cells were pelleted, resuspended and the effects of the various antisera on proliferating cells assessed through the ability of the remaining cells to incorporate H3-thymidine into acid insoluble polydeoxyribonucleotides. The effects of the various antisera on Tk cells generated either in vitro or in vivo during skin graft rejection were assessed through the ability of the cells remaining after treatment with antiserum plus complement to lyse Cr51-labeled target cells. Only those cells that expressed Ia markers could stimulate responses to H-2K, D and/or I region antigens in mixed lymphoid cell cultures, even though all lymphoid cells apparently express the H-2K and H-2D-coded private antigens. Ia markers, therefore, distinguished the subset of cells, which includes as its members the stimulating cells. The Ia marker was expressed on the cell membrane of at least one of the two T cell subsets that collaborate in the development of T effector cells to H-2-associated alloantigens. The cells remaining after lysis with the antiserum plus complement could no longer respond in the mixed lymphocyte reaction. Syngeneic non-T cells could not reconstitute the response. Most activated and proliferating cells expressed the Ia determinant(s) but a proportion of T effector or killer cells did not. These results suggest that the Ia specificity may be expressed transiently during the activation, proliferation and differentiation of a lymphoid cell into a specific mature effector cell.

53. GENETIC REGULATION OF THE ANTIBODY RESPONSE TO H-2DB ALLOANTIGENS IN MICE. I. DIFFERENCES IN ACTIVATION OF HELPER T CELLS IN C57BL/10 AND BALB/C CONGENIC STRAINS.

Wernet D, Lilly F
Albert Einstein Coll. Med., Bronx, N. Y.
J Exp Med; 141(3):573-583 1975

C57BL/10, B10 and A.BY mice were used in order to determine if the altered antibody response of C57BL/10 mice was associated within or outside of the H-2 complex. Lymphocyte- mediated cytolysis was measured using peritoneal exudate cells of immunized mice and target cells of C57BL leukemia or phytohemagglutinin-induced blast cells. Normal T-cell-mediated cytotoxicity to H-2Db tumor cells was seen in B10.A(5R) mice immunized with C57BL/10 spleen cells, but no antibody response to H-2Db alloantigens was seen. An IgG response was seen in B10.A (5R) mice immunized with A.BY or B10.A (2R) cells. The B10.A (2R) cells differ from B10.A (5R) cells at H-2Db, H-2Kk and H-2Ik cell surface antigens. C57BL/10 mice immunized with congenic H-2g cells made only IgM antibodies, whereas BALB/c mice made IgG antibodies. T-helper cells failed to switch from IgM to IgG when the only differences on the immunizing cells were the H-2Db alloantigens. The results indicate that T-cell helper function is regulated by genes separate from H-2, and that T-cell helper function is regulated by different genes from those that regulate T-cell mediated cytotoxicity.

54. SUPPRESSOR T CELLS: CLASSES OF ACTIVITY. (PP. 1-19)

Gershon RK
Immune Depression and Cancer Proceedings of the Second Irwin Strasburger Memorial Seminar on Immunology. Department of Medicine, Cornell University, Medical College, The New York Hospital, New York NY, 1975.

The evidence on suppressor T-cells is classified and reviewed in order to further the understanding of the immunoregulation by T-cells. Both direct and indirect types of immunological tolerance are considered: the latter is T-cell dependence of specific B-cell unresponsiveness while the former concerns suppression by T-cells. Antigenic competition is discussed in relation to mitogen activated T-cells, cultured T-cells, and spleen seeking T-cells. Studies have indicated a prophylactic role by T-cells in the prevention of autoimmune disease. Particularly, T-cell depletion may hasten and increase the appearance of auto-antibodies and autoimmune diseases in some strains of mice, rats, and chickens. Class specific T-cell suppression, augmentation of thymus independent immune responses, and the control of genetically determined immunological unresponsiveness are discussed. Feedback regulation from both T- and B- cells is considered. Regulation of non-antigen induced events such as allotype suppression, and immunoglobulin production are presented.

55. SUPPRESSIVE INTERACTIONS BETWEEN DIFFERENT MLR LOCI. (PP. 193-202)

Gershon RK
The Role of Products of the Histocompatibility Gene Complex in Immune Responses. Katz DH, Benacerraf B, ed. New York, Academic Press Inc, 1976.

The reactions of different mouse responder T cells to foreign cells which differ either at the M locus, the H-2 locus, or the two loci together were examined. The DNA synthetic response of lymph node T cells to foreign M loci was inhibited by the presence of foreign H-2 loci on stimulator cells. This inhibition could be overcome by pulsing the responding cells with either cortisone or cycloheximide. Splenic T cells were not inhibited. In light of present knowledge of the H-2 locus and T cell subsets these data are interpreted as follows: Ly-2+, 3+ cells, activated by H-2D or H-2K determinants act as the suppressors and that the Ly-1+, 2+, 3+ splenic T cells either prevent the suppressive interaction or obscure it by making a strong positive response of their own. (20 refs)

56. GENETIC CONTROL OF IMMUNE RESPONSES IN VITRO. V. STIMULATION OF SUPPRESSOR T CELLS IN NONRESPONDER MICE BY THE TERPOLYMER L-GLUTAMIC ACID^{60}-L-ALANINE^{30}-L-TYROSINE^{10} (GAT).

Kapp JA, Pierce CW, Schlossman S, Benacerraf B
Dept. Pathology, Harvard Medical Sch., Boston, MA, 02115
J Exp Med; 140:648-659 1974

In an effort to elucidate the mechanism involved in tolerance induced by the random terpolymer L-glutamic acid^{60}-L-alanine^{30}-L-tyrosine^{10} (GAT) in nonresponder mice, the immunocompetence of T and B cells from spleens of nonresponder mice, previously rendered unresponsive by injection of GAT, and the effects of such populations of T and B cells on the development of GAT-specific plaque-forming cell (PFC) responses by normal nonresponder spleen cells incubated with GAT complexed to methylated bovine serum albumin (MBSA) were investigated. Treatment of nonresponder DBA/1 mice with GAT rendered their spleen cells unable to develop a GAT-specific response when incubated with GAT-MBSA, although their response to sheep RBC was equivalent to that of spleen cells from normal DBA/1 mice. B cells from GAT-primed mice could respond to GAT-MBSA in vitro in the presence of added carrier-primed (GAT-MBSA) helper T cells. Spleen cells from GAT-primed DBA/1 mice specifically suppressed the development of GAT-specific PFC responses by spleen cells from normal DBA/1 mice incubated with GAT-MBSA. The activity of suppressor T cells induced by GAT was antigen specific. Models for the mechanism and site of action of suppressor T cells in the GAT system are discussed. (18 Refs)

57. GENETIC CONTROL OF SPECIFIC IMMUNE SUPPRESSION. II. H-2-LINKED DOMINANT GENETIC CONTROL OF IMMUNE SUPPRESSION BY THE RANDOM COPOLYMER L-GLUTAMIC ACID^{50}-L-TYROSINE^{50} (GT).

Debre P, Kapp JA, Dorf ME, Benacerraf B
Dept. Pathology, Harvard Medical Sch., Boston, MA, 02115
J Exp Med; 142:1447-1454 1975

Mice of various strains were preimmunized with the random copolymer L-glutamic acid- (50%) L-tyrosine (50%) (GT), and tested for their response to GT conjugated with methylated bovine serum albumin (MBSA) mixed with complete Freund's adjuvant. Antibody responses were measured by an indirect hemolytic plaque assay for IgG-specific plaque-forming cells. GT preimmunization was found to depress response of some strains of inbred mice to challenge with GT-MBSA, but not others. Hybridization demonstrated that GT-specific suppression is inherited as a dominant trait. Testing of congenic strains of mice showed that GT nonresponse was related to their histocompatibility-2 (H-2) haplotype but not to their background genotype. Congenic strains bearing H-2**d or H-2**s, but not H-2'a or H-2**b, were suppressed by GT. Testing of mice with GT and the cross-reacting terpolymer, L-glutamic acid-L-alanine-L-tyrosine, showed that the pattern and specificity of immune suppression for the copolymers are distinct in different strains. This trait was considered to be under the control of immune suppression gene(s) which are coded for in the H-2 complex. The immune response is discussed. (15 Refs)

58. GENETIC CONTROL OF SPECIFIC IMMUNE SUPPRESSION. III. MAPPING OF H-2 COMPLEX COMPLEMENTING GENES CONTROLLING IMMUNE SUPPRESSION BY THE RANDOM COPOLYMER L-GLUTAMIC ACID^{50}-L-TYROSINE^{50} (GT).

Debre P, Waltenbaugh C, Dorf M, Benacerraf B
Dept. Pathology, Harvard Medical Sch., Boston, MA, 02115
J Exp Med; 144:272-276 1976

Various inbred, congenic resistant and histocompatibility-2 (H-2) mouse strains were tested for their ability to show suppressed response to a hapten conjugate of the copolymer L-glutamic acid-L-tyrosine (GT) (50:50 M ratio) with methylated bovine serum albumin (MBSA) after preimmunization with GT. Strains bearing H-2**k, H-2**d, or H-2**s but not H-2**b or H-2**a were specifically suppressed by GT preimmunization. Tests with recombinant strains showed that this suppression is controlled by at least two complementing or interacting loci which may be restricted when the genes come from different haplotypes, and that the genes for the different suppressor haplotypes are different. The D and K loci were found not to be responsible for the suppressor activity, but the I and/or S regions, more probably the I, are responsible. Complementing genes, designated immune suppressor (Is) genes, in I-C and genes in I-A and/or I-B are responsible for the development of GT-specific suppression. The relationship between Is and immune response (Ir) genes is discussed. (12 Refs)

C. The Histocompatibility Gene Products as Mediators of Immune Cell Interaction

59. FUNCTIONAL ANALYSIS OF IA ANTIGENS IN RELATION TO GENETIC CONTROL OF THE IMMUNE RESPONSE. (PP. 257-282)

McDevitt HO
The Role of Products of the Histocompatibility Gene Complex in Immune Responses. Katz DH, Benacerraf B, ed. New York, Academic Press Inc, 1976.

The Ia antigens in relation to the genetic control of the immune response are analyzed functionally. The distribution of Ia antigens on T cell and B cell subpopulations correlates with the T cell subtypes involved in regulating the immunoglobulin (Ig)G antibody response and with the B cell types expressing that response. The presence of Ia antigens on suppressor T cells raises problems. Although it has been assumed that Ia antigens on any cell type are produced by that cell type, this assumption requires verification. The demonstration of Ia antigens in suppressor T cell factors and on suppressor T cells is compatible with the fact that antigen-specific suppression is under H-2 linked genetic control. Because antigen-induced suppression may be a transient phenomenon, it may function as a mechanism to regulate and limit the B cell response to a specific antigen. It is concluded that the Ia antigens are involved in the induction of B cells to switch from IgM to IgG antibody production.

60. EXPRESSION OF IA DETERMINANTS ON IMMUNOCOMPETENT CELLS.

Hammerling GJ, Eichmann K
Institut fur Genetik der Universitat zu Koln, Weyertal 121, D-5000 Koln 41, W. Germany
Eur J Immunol; 6(8):565-569 1976

The expression of Ia (immune response region-associated) antigens on the surface of CBA/J mouse lymphocyte subpopulations with defined immunological functions was investigated by negative selection of sub- populations with anti-Ia sera and complement. Ia determinants were found on both unprimed (IgM) and on primed (IgG) antibody-forming B precursor cells. No Ia antigens were detected on the surface of helper T cells. The experiment suggests that the small fraction of Ig-positive and Ia- negative cells in murine spleens are immunologically immature because they are not able to respond significantly to a primary antigenic challenge. Ia determinants apparently must be expressed first on the surface of B cells before effective cooperation with T cells and macrophages can take place. In contrast, suppressor T cells were sensitive to treatment with anti-Ia sera and complement; this demonstrates the presence of Ia determinants on this T cell subpopulation. (26 refs)

61. T LYMPHOCYTE-ENRICHED MURINE PERITONEAL EXUDATE CELLS. III. INHIBITION OF ANTIGEN-INDUCED T LYMPHOCYTE PROLIFERATION WITH ANTI-IA ANTISERA.

Schwartz RH, David CS, Sachs DH, Paul WE
Natl. Cancer Inst., Natl. Inst. Health, Bethesda, MD 20014
J Immunol; 117(2):531-540 1976

The effect of anti-histocompatibility antisera on the proliferative response was investigated. The mouse strains used were A.TH, A.TL, A.AL(Thy 1.1), A.AL (Thy 1.2), HTH/AoSF and HTI/BoySf. The continuous presence of anti-Ia antisera in the cultures inhibited the responses to the antigens poly (Glu^{58} Lys^{38} Tyr^{4}) GLT, poly (Tyr,[illegible] poly D,L Ala - poly Lys (T,G)-A - L, poly (Phe,Glu)-poly D,L Ala - poly Lys (phi,G)-A - L, lactate dehydrogenase H4 , staphylococcal nuclease, and the IgA myeloma protein, TEPC 15. The T lymphocyte proliferative responses to all of these antigens was under the genetic control of major histocompatibility-linked immune response genes. The anti-Ia antisera were also capable of inhibiting proliferative responses to antigens such as purified protein derivative of tuberculin (P-PD), to which all strains respond. In contrast, antisera directed solely against H-2K or H-2D antigens did not give significant inhibition. Anti-Ia antisera, capable of reacting with antigens coded for by genetically defined subregions of the I locus, were capable of completely inhibiting the proliferative response. In GLT and (T,G)-A - L, an Ir gene controlling the T lymphocyte proliferative response to the antigen had been previously mapped to the same subregion as that which coded for the Ia antigens recognized by the blocking antisera. Finally, in F1 hybrids between responder and nonresponder strains, the anti-Ia antisera showed haplotype-specific inhibition. That is, anti-Ia antisera, directed against the responder haplotype, could completely block the antigen response controlled by Ir genes of that haplotype; anti-Ia antisera, directed against Ia antigens of the nonresponder haplotype, gave only partial or no inhibition. Since this selective inhibition was reciprocal, depending on which antigen was used, it suggested that the mechanism of anti-Ia antisera inhibition was not cell killing or a nonspecific turning off of the cell but rather a blockade of antigen stimulation at the cell surface. The selective inhibition demonstrates a phenotypic linkage between Ir gene products and Ia antigens at the cell surface. These results, coupled with the known genetic linkage of Ir genes and the genes coding for Ia antigens, suggest that Ia antigens are determinants on Ir gene products.

62. CELL-MEDIATED LYMPHOLYSIS OF TRINITROPHENYL-MODIFIED AUTOLOGOUS LYMPHOCYTES. CONFIRMATION OF GENETIC CONTROL OF RESPONSE TO TRINITROPHENYL-MODIFIED H-2 ANTIGENS BY THE USE OF ANTI-H-2 AND ANTI-IA ANTIBODIES.

Schmitt-Verhulst AM, Sachs DH, Shearer GM
Natl. Cancer Inst., Bethesda, Md. 20014
J Exp Med; 143(1):211-217 1976

Splenic lymphocytes from B10.A and B10.D2 mice were sensitized in vitro to trinitrophenyl (TNP)-modified autologous spleen cells. The effector cells generated were assayed in a ^{51}Cr-release assay on TNP-modified syngeneic or congenic spleen target cells. Effector cells from B10.A donors lysed TNP-modified H-2Kk- but not H-2Dd-region products, whereas B10.D2 effectors reacted with modified products of both the H-2Kd and H-2Dd regions. As an independent confirmation that this selective K-end lysis by B10.A effector cells is due to an H-2-linked responder cell defect, anti-H-2Kk but not anti-H-2Dd sera inhibited the lysis of B10.A-TNP targets by B10.A effectors. In contrast, anti-H-2Dd sera inhibited the lysis of B10.A-TNP targets by B10.D2 effectors. Anti-Ia antibodies had no detectable effect on lysis. Anti-TNP-keyhole limpet hemocyanin sera blocked the lysis of TNP-modified targets, irrespective of whether the effector cells were directed against TNP-modified autologous H-2 products or H-2 alloantigens. These results independently verify that B10.A responding lymphocytes do not generate effector cells to TNP-modified H-2Dd products, whereas B10.D2 lymphocytes do, and suggest that some TNP groups are sterically close to (or part of) the serologically defined H-2K- and H-2D-region antigens.

63. THE ROLE OF HISTOCOMPATIBILITY GENE PRODUCTS IN COOPERATIVE CELL INTERACTIONS BETWEEN T AND B LYMPHOCYTES. (PP. 569-596)

Katz DH, Benacerraf B

Dept. Pathology, Harvard Medical Sch., Boston, MA, 02115

The Immune System: Genes, Receptors, Signals. Sercarz EE, Williamson AR, Fox CF, ed. New York, Academic Press, 1974.

Three different experimental approaches are presented which support the hypothesis that the active regulation of B cell responses, in T-B cell cooperation, is through interaction with soluble mediators produced and released by T cells. These approaches are as follows: the identification of genes and gene products in the major histocompatibility gene complex and the role of their products in governing optimal T-B cell cooperative interaction; the biological and biochemical properties of a non-specific mediator produced by populations of activated T cells and its activity on the in vitro antibody responses of mouse lymphocytes; and the elicitation of hapten-specific antibody responses in vivo by stimulation of carrier-specific T cells and hapten-specific B cells by their relevant determinants administered independently and on separate molecules. The existence on the B lymphocyte surface membrane of a site closely related to the histocompatibility specificity that is critically involved in physiologic T-B cell interaction is postulated. The site is envisaged as an 'acceptor' molecule either for the active T cell product or the T cell itself. Immune response genes may be expressed in B cells as well as T cells. Their activity is concerned with successful T-B cell interactions in specific immune responses involving carrier recognition.

64. CELL INTERACTIONS BETWEEN HISTOINCOMPATIBLE T AND B LYMPHOCYTES. II. FAILURE OF PHYSIOLOGIC COOPERATIVE INTERACTIONS BETWEEN T AND B LYMPHOCYTES FROM ALLOGENEIC DONOR STRAINS IN HUMORAL RESPONSE TO HAPTEN-PROTEIN CONJUGATES.

Katz DH, Hamaoka T, Benacerraf B

Dept. Pathology, Harvard Medical Sch., Boston, MA, 02115

J Exp Med; 137:1405-1418 1973

Antigen-specific interactions in which carrier-primed T cells provide stimulation for B cells to respond to hapten-protein conjugates were studied using histoincompatible T and B cells under conditions that avoid the allogeneic effect. Spleen cells from either bovine gamma globulin (BGG)-primed or normal parental donor mice were injected iv into F1 (BALB/c x A/J) mice, which were irradiated 24 hr later and injected with dinitrophenyl- (DNP) keyhole limpet hemocyanin-(KLH) primed anti-theta serum plus complement-treated spleen cells (B cells). Mice were then challenged ip with DNP-BGG. Syngeneic T and B cells from either strain cooperated, but allogeneic lymphocytes did not. This was confirmed in vitro using either DNP-KLH-primed BALB/c spleen cells cultured with syngeneic or allogeneic KLH-primed T lymphocytes in the presence of DNP-KLH, or by using spleen cells from DNP-BGG-primed A/J mice with syngeneic or allogeneic KLH-primed T cells in the presence of DNP-BGG. Antigen-specific cell interactions were also shown to occur between parental T and F1 B lymphocytes, and vice versa. Possible reasons for failure of allogeneic T and B cells to form cooperative interactions are discussed. (15 Refs)

65. CELL INTERACTIONS BETWEEN HISTOINCOMPATIBLE T AND B LYMPHOCYTES. IV. INVOLVEMENT OF THE IMMUNE RESPONSE (IR) GENE IN THE CONTROL OF LYMPHOCYTE INTERACTIONS IN RESPONSES CONTROLLED BY THE GENE.

Katz DH, Hamaoka T, Dorf ME, Maurer PH, Benacerraf B

Dept. Pathology, Harvard Medical Sch., Boston, MA, 02115

J Exp Med; 138:734-739 1973

Antigen-specific T-B cell cooperative interactions were studied using T cells primed to the terpolymer L-glutamic acid-L-lysine-L-tyrosine (GLT) from F1 hybrids (which are responders to this carrier molecule) of BALB/c (responders) and A/J (nonresponders) mice. GLT was conjugated with the hapten dinitrophenyl (DNP). DNP-bovine gamma globulin (BGG), to which both strains of mice respond, was also tested. It was shown that both BALB/c and A/J DNP-specific B cells interacted effectively with F1 BGG-specific T cells. Only B cells from BALB/c mice cooperated with F1 GLT-primed T cells. B cells from A/J mice did not interact. Disparity at the immune response (Ir) locus, which controls response to GLT, is at least one factor responsible for preventing effective T-B cell interactions. The Ir gene and its relation to the major histocompatibility complex are discussed. (8 Refs)

66. HISTOCOMPATIBILITY GENE PRODUCTS AS MEDIATORS OF LYMPHOCYTE INTERACTIONS. (PP. 541-552)

Katz DH, Armerding D, Eshhar Z

The Role of Products of the Histocompatibility Gene Complex in Immune Responses. Katz DH, Benacerraf B, ed. New York, Academic Press Inc, 1976.

The histocompatibility gene products as mediators of lymphocyte interactions are described. The molecules involved in the regulatory cell interactions in lymphocyte differentiation and immune responses are gene products of the major histocompatibility complex; they are, in particular, the gene products of the I region of the mouse H-2 complex. Biologically active molecules, obtained in soluble form from T lymphocytes and possessing Ia determinants, exert their functional effects in either antigen-specific or non-specific ways. A class of molecules is produced by antigen-activated T cells, exerts biological activity in the regulation of immune responses, and bears determinants coded for by I region genes. The functional effects of these molecules are either suppressive or enhancing, and they seem to be capable of acting on B lymphocytes and on T lymphocytes. It is concluded that more must be learned about the soluble T cell factors.

67. MACROPHAGE-LYMPHOCYTE INTERACTION AND ANTIGEN RECOGNITION.

Rosenthal AS, Shevach EM

Lab. Clinical Investigation, Natl. Inst. Allergy and Infectious Diseases, NIH, Bethesda, MD, 20014

Fed Proc; 34(8):1743-1748 1975

The functional and morphologic aspects of immunologically relevant macrophage-lymphocyte interaction have been characterized in inbred guinea pigs. Using an in vitro assay of lymphocyte binding to macrophage monolayers, what appear to be sequential events in macrophage-lymphocyte interaction have been characterized. The initial event is an antigen-independent interaction between macrophage and lymphocyte whose characteristics are rapid reversibility, species but not strain specificity, and dependence on macrophage metabolism. If macrophages bear antigen for which a given lymphocyte possesses a specific receptor, then a second nonreversible antigen-dependent stabilization of binding occurs. This latter step requires that macrophage and lymphocyte be histocompatible. These observations parallel functional studies of the histocompatibility dependence of macrophage-associated antigen initiation of lymphocyte DNA synthesis in guinea pigs. A model sequence of the physical events correlating with functional macrophage-lymphocyte interaction is proposed.

68. FUNCTION OF MACROPHAGES IN ANTIGEN RECOGNITION BY GUINEA PIG T LYMPHOCYTES. I. REQUIREMENT FOR HISTOCOMPATIBLE MACROPHAGES AND LYMPHOCYTES.

Rosenthal AS, Shevach EM
Lab. Clinical Investigation, Natl. Inst. Allergy and Infectious Diseases, NIH, Bethesda, MD, 20014
J Exp Med; 138:1194-1212 1973

The importance of macrophage and T lymphocyte histocompatibility determinants in the expression of antigen recognition was evaluated in strain 2 and strain 13 guinea pigs (Division of Research Services, NIH). Initial studies examined the ability of strain 2 and 13 macrophages to activate immune strain 2 and 13 peritoneal exudate lymphocytes (PEL). The antigen-dependent incorporation of tritiated thymidine into new DNA was assessed at 72 hr. Strain 2 macrophage-associated antigen initiated immune strain 2 PEL DNA synthesis quite effectively (189,390 cpm vs control 1,250 cpm). The same macrophages mixed with immune strain 13 PEL stimulated little DNA synthesis (13,540 cpm). Similarly, antigen-pulsed strain 13 macrophages induced little DNA synthesis when mixed with immune strain 2 PEL, but stimulated considerable DNA synthesis when mixed with immune strain 13 macrophages (58,700 cpm vs control 650 cpm). The ability of (2 x 13)F1 lymph node lymphocytes (LNL) to respond to parental macrophages and the ability of F1 macrophages to activate parental LNL confirmed the observation that macrophage-rich peritoneal exudate cells pulsed with antigen efficiently activate only syngeneic lymphocytes. The failure of allogeneic macrophages to effectively initiate immune lymphocyte proliferation was not the result of the presence of an inhibitor of blastogenesis released in mixtures of allogeneic cells, peculiarities of the antigen or lymphoid cells employed, nor differing kinetics of activation by allogeneic macrophages. (36 Refs)

69. FUNCTION OF MACROPHAGES IN ANTIGEN RECOGNITION BY GUINEA PIG T LYMPHOCYTES. II. ROLE OF THE MACROPHAGE IN THE REGULATION OF GENETIC CONTROL OF THE IMMUNE RESPONSE.

Sheyach EM, Rosenthal AS
Lab. Immunology, Natl. Inst. Allergy and Infectious Diseases, NIH, Bethesda, MD, 20014
J Exp Med; 138:1213-1229 1973

A study was made of the ability of macrophages obtained from a parental guinea pig to activate F1 T lymphocyte proliferation to an antigen, the response to which is controlled by an immune response (Ir) gene absent in the parent. Whether the inhibitory effect exerted by alloantisera was a nonspecific block of macrophage-T lymphocyte interaction, a specific block of the product of Ir genes, or both, was also studied. Parental macrophages, pulsed with an antigen, were mixed with T cells derived from the (nonresponder x responder) F1 and the resultant stimulation was measured. No stimulation was noted when column-purified F1 lymph node lymphocytes were mixed with antigen-pulsed macrophages from the nonresponder parent. However, when the highly reactive peritoneal exudate lymphocyte population was used as indicator cells, parental macrophages pulsed with an antigen whose Ir gene they lacked were capable of initiating F1 T cell proliferation. The magnitude of stimulation was approx 1/10 that seen when macrophages from either the responder parent or the F1 were used. Antigen recognition sites on the T lymphocyte are hypothesized as being physically related to a macrophage-binding site and both as being linked to the serologically determined histocompatibility antigens. (14 Refs)

70. THE FUNCTION OF MACROPHAGES IN ANTIGEN RECOGNITION BY GUINEA PIG T LYMPHOCYTES. III. GENETIC ANALYSIS OF THE ANTIGENS MEDIATING MACROPHAGE-T LYMPHOCYTE INTERACTION.

Shevach EM
Lab. Immunology, Natl. Inst. Allergy and Infectious Diseases, NIH, Bethesda, MD, 20014
J Immunol; 116(5):1482-1489 1976

The possibility that macrophage-T lymphocyte interaction is mediated by the immune response-associated (Ia) antigens of the guinea pig major histocompatibility complex (MHC) was investigated for the purpose of gathering support for the concept of the functional expression of the Ir gene product in the macrophage. Experiments using combinations of macrophages and lymphocytes derived from animals that differed by either B region antigens or Ia antigens demonstrated that in general the Ia antigens or the products of closely linked genes mediate macrophage-T lymphocyte interaction. Combinations of macrophages and T cells derived from outbred animals with different Ia antigens and/or Ir gene products revealed that sharing of Ir gene products between macrophages and T cells was not sufficient for effective macrophage-T lymphocyte interaction. (29 Refs)

III. NATURAL KILLER CELLS

71. RESISTANCE OF H-2 HETEROZYGOUS MICE TO PARENTAL TUMORS. I. HYBRID RESISTANCE AND NATURAL CYTOTOXICITY TO EL-4 ARE CONTROLLED BY THE H-2D-HH-1 REGION.

Harmon RC, Clark EA, O'Toole C, Wicker LS
Dept. Microbiology, Center for Health Sciences, Univ. California at Los Angeles, Los Angeles, CA 90024
Immunogenetics; 4(6):601-607 1977

Control of hybrid resistance to the C57BL lymphoma EL-4 by the H-2 chromosomal region was studied. Heterozygosity at defined portions of the H-2 complex was achieved by mating C57BL/10, B10. A, and congenic H-2a/H-2b recombinant mice to produce F-1 recipients for the tumors. It was established that resistance to the EL-4 tumor was provided by heterozygosity within or near the H-2D-Hh-1 region of the genome. Spleen cell-mediated natural cytotoxicity was determined by Cr-51 release assay, and it was found that EL-4 resistant H-2Db/H-2Dd heterozygous mice have a significantly greater spleen cell-mediated cytotoxicity against EL-4 target cells than do homozygous H-2Db mice. All other heterozygous strains had significant increases in cell-mediated cytotoxicity to the target tumor cells than did the parent lines. The results indicate that the Hh-1 products expressed in the parental H-2Db cells were not manifested as a dominant character in the H-2Db/H2Dd F-1 hybrid cells. (21 Refs)

72. RESISTANCE OF H-2 HETEROZYGOUS MICE TO PARENTAL TUMORS. II. CHARACTERIZATION OF HH-1-CONTROLLED HYBRID RESISTANCE TO SYNGENEIC FIBROSARCOMAS AND THE EL-4 LYMPHOMA.

Clark EA, Harmon RC, Wicker LS
Immunogenetics Group, Dept. Microbiology and Immunology, Center for the Health Sciences, Univ. California, Los Angeles, CA 90024
J Immunol; 119(2):648-656 1977

Evidence that Hh-1 chromosome locus controls hybrid resistance to two 3-methylcholanthrene-induced B10.A(4R) fibrosarcomas as well as C57BL lymphoma EL-4 in H-2 heterozygous mice is presented. Evidence suggests that Hh-1 maps within the H-2 complex between the S and D regions. Other H-2 genes influence fibrosarcoma growth in histocompatible hosts and are mapped within the I region. Hh-1-controlled hybrid resistance was clearly expressed when tumor cells were inoculated into the peritoneal cavity, but not expressed when inoculated subcutaneously. This resistance was expressed against a normal cell constituent coded for or regulated by the Hh-coded locus. Hh-1 codes for or regulates the expression of cell surface antigens found on normal H-2b parental cells. The polygenic control of the immune response to fibrosarcomas is apparently influenced by several loci within the H-2 complex. (42 Refs)

73. RESISTANCE OF IRRADIATED MICE TO LYMPHOMA TRANSPLANTS: IMPORTANCE OF THE HH-1/D REGION OF THE H-2 COMPLEX AND PREFERENTIAL EXPRESSION IN THE SPLEEN. (PP. 373-375)

Cudkowicz G, Riccardi C
Immuno-aspects of the Spleen, Proceedings of a Conference on Immunodynamics, Cleveland, Ohio, May 19 and 20, 1976. Battisto JR, Streilein JW, ed. New York, North-Holland Publishing Co., 1976.

The genetic differences responsible for the localized resistance of irradiated mice to lymphoma transplants were found to reside in the Hh-1/D region of the H-2 complex. Differences at other regions were inconsequential for lymphoma growth in irradiated hosts. The administration of silica particles rapidly abrogated resistance in both euthymic and athymic animals. An in vitro model of resistance was developed in which F1 lymphocytes generated cell-mediated cytotoxic activity specific for parental spleen and lymphoma cells. The results of these in vitro and in vivo experiments pointed to a resemblance to hemopoietic histocompatibility (the rejection of foreign bone marrow grafts by irradiated mice), and they indicated that the products of Hh genes are expressed in lymphoma cells. Mechanisms of anti-Hh responses may be of significance in tumor immunity. (no Refs)

74. NATURAL KILLER CELLS IN THE MOUSE. (PP. 77-85)

Kiessling R
The Role of Products of the Histocompatibility Gene Complex in Immune Responses. Katz DH, Benacerraf B, ed. New York, Academic Press, 1976.

Present knowledge concerning the nature, genetic regulation, and in vivo relevance of natural killer cells (NKC) identified in certain strains of mice is summarized. These nonimmune lymphocytes have been demonstrated to have the capacity to kill certain syngeneic, semisyngeneic, and allogeneic lymphoma cells in a ^{51}Cr assay. While cytolytic activity is selective, the exact nature of the specificity is unknown. A pronounced age dependence of the NKC has been observed, with peak activity in lymphocytes from mice of approx 2 mo of age and little if any activity in lymphocytes from newborn or older mice. Lymphocytes from spleen and peripheral blood had the highest activity, those from lymph nodes and bone marrow had somewhat less activity, and thymus cells were totally inactive. Various cell fractionation procedures have indicated that the NKC are neither T nor B lymphocytes, nor do they share the properties of the K cells of antibody-dependent cellular cytotoxicity. Hybridization studies using mouse strains of low and high activity have indicated that reactivity is dominant, and backcross experiments have indicated a strong H-2 linked factor. A high degree of agreement between in vitro cytolytic activity and in vivo tumor resistance has been observed in different genotypes using a Moloney virus induced lymphoma. In vivo resistance to the same lymphoma appeared to be H2-linked and thymus-independent. (18 refs)

75. NON-T-CELL RESISTANCE AGAINST A MOUSE MOLONEY LYMPHOMA.

Kiessling R, Petranyi G, Klein G, Wigzell H
Dept. Tumor Biology, Karolinska Inst., 104 01 Stockholm 60, Sweden
Int J Cancer; 17(2):275-281 1976

In vivo rejection studies with a Moloney lymphoma line in various normal semisyngeneic F1 hybrid mice showed a clear positive correlation between in vitro natural cytotoxicity and in vivo rejection. Thus mice could be grouped as high- or low-reactive according to their in vitro cytolytic behavior as well as their in vivo rejection potential. A lymphoid cell without detectable T- or B-cell markers was responsible for the in vitro killing effect. The in vivo growth inhibitory function was T-cell-independent. Lymphoid cells depleted of T- and B-cells and transferred together with Moloney lymphoma cells into an irradiated syngeneic recipient were highly efficient in delaying tumor growth. Furthermore, syngeneic or semisyngeneic thymectomized, irradiated, fetal-liver-reconstituted mice showed an increased in vivo resistance to a Moloney lymphoma compared to control mice. In contrast, tumor cells histoincompatible with regard to the H-2 locus showed the expected preferential growth in the thymectomized animals. It is concluded that the major in vivo resistance against transplantation of the syngeneic or semisyngeneic Moloney lymphoma used in this study is T-independent.

76. THE GENETIC CONTROL OF NATURAL KILLER CELL ACTIVITY AND ITS ASSOCIATION WITH IN VIVO RESISTANCE AGAINST A MOLONEY LYMPHOMA ISOGRAFT.

Petranyi GG, Kiessling R, Povey S, Klein G, Herzenberg L, Wigzell H
Dept. Tumor Biology, Karolinska Inst., 104 01 Stockholm 60, Sweden
Immunogenetics; 3(1):15-28 1976

The possible in vivo role of natural killer spleen cells against Moloney lymphoma line YAC-1 (strain A origin) was examined in host mice of several genotypes. Spleen lymphocytes of A mice have low natural killing activity, while those of C57BL have high activity. The in vitro killer activity of spleen cells correlated with in vivo host resistance to YAC lymphoma. In vivo and in vitro tests were carried out on (A x C57BL)F1 x A backcross mice. The natural in vitro killer activity appeared to be under polygenic control, including a strong H-2-linked factor. No linkage was seen with five different isozyme loci, with the Ig-1 locus, or with C5 serum activity. In vivo resistance showed strong linkage with the H-2 complex. A weak linkage was found with the coat color locus C in (A x C57BL)F1 x A backcross mice. In the same backcross mice, there was a correlation between in vitro killer activity and in vivo resistance In vivo resistance was particularly strong in mice that combined the H-2b-linked resistance factor with a high cytolytic activity in vitro. (34 refs)

77. IMMUNE INHIBITION OF ALLOGENEIC LYMPHOMA CELLS IN THE PERITONEAL CAVITY OF MICE.

Fioretti MC, Liberati M, Bonmassar E, Cudkowicz G
Lab. Tumor Immunol. Chemother., Univ. Perugia, Perugia, Italy
Cancer Res ; 35(1):30-36 1975

Inbred mice of both sexes of the congenic-resistant strains C57BL/10ScSn (B10, H-2b), B10.A(H-2a), B10.A(5R)(H-2i), and BALB/c xDBA/2)F1 hybrids were sensitized with cells from normal spleens, transplantable syngeneic lymphomas (LAF-17 and L5MF-22), or allogeneic lymphomas (L1210/Ha and L5178Y) differing for alloantigens specified by the major histocompatibility complex. The allograft reactivity was determined in these mice from 3-11 d later by the disappearance of inj lymphoma cells from the peritoneal cavity or by the inhibition of DNA synthesis. For the disappearance test, target cells were labeled with ^{125} I-5-iododeoxyuridine before transfer. For the inhibition test, unlabeled cells were transferred and subsequently exposed to the DNA precursor, ^{125} Iododeoxyuridine. Both tests were immunogenetically specific in detecting secondary allograft reactions, but the disappearance test was less sensitive. By inhibition of DNA synthesis, both primary and secondary (3-8 d later) reactions were detected. Alloantigens associated with the H-2K-Ir regions of Murine Linkage Group IX were more immunogeneic than those associated with the Ss-H-2D-Tla regions in eliciting antilymphoma reactions, with female mice responding better than male mice. Procedures used to assess the level and specificity of allograft reactions in the peritoneal cavity of mice bearing syngeneic or allogeneic tumors in other sites were compatible with survival of test animals. The peritoneal inhibition test was sensitive enough to monitor transplantation immunity in vivo and could be applied to animals bearing tumors in sites other than the peritoneum and undergoing chemotherapy. (15 refs)

78. LYMPHOMA INDUCED BY HERPESVIRUS: RESISTANCE ASSOCIATED WITH A MAJOR HISTOCOMPATIBILITY GENE.

Longenecker BM, Pazderka F, Gavora JS, Spencer JL, Ruth RF
Department of Immunology, University of Alberta, Edmonton, Alberta, Canada T6G 2E9
Immunogenetics; 3(4):401-407 1976

Studies with crosses of inbred chicken lines demonstrate that resistance to Marek's disease, a herpesvirus-induced lymphoma of chickens, is associated with an allele (B^{21}) of the major histocompatibility locus (the B locus). The B^{21} allele is thus the first genetic marker for resistance to herpesvirus-induced neoplastic disease, and our studies suggest the means whereby similar associations might be found in man. (Author Abstract)

79. INDUCTION OF F1 HYBRID ANTIPARENT CYTOTOXIC EFFECTOR CELLS: AN IN VITRO MODEL FOR HEMOPOIETIC HISTOINCOMPATIBILITY.

Shearer GM, Cudkowicz G
Immunol. Branch, NCI, Bethesda, Md.
Science; 190(4217):890-893 1975

The generation of primary F1 antiparent-specific cytotoxic reactions in spleen cells cultures is reported. Cytotoxicity was assessed against transplantable ascites tumors sharing the H-2 type of the stimulating cells sharing the H-2D-Hh-1 region. Effector cells cytotoxic for EL-4 did not lyse cells of different H-2D-Hh-1 type. F1 responding cells did not generate cytotoxic effectors for EL-4 when stimulated with parental cells of H-2d and H-2a type. The antigenic differences detected are controlled by the H-2D-Hh-1 region of the murine major histocompatibility complex. The parent-F1 combinations demonstrating F1 antiparent activity in vitro are the same as those demonstrating F1 rejection of parental hemopoietic grafts in vivo. Hence, the F1 antiparent cytotoxicity test serves as an in vitro model for the recognition and effector phases of hybrid resistance to parental hemotopoietic grafts and may be of value in clinical transplantation. (14 refs)

IV. ROLE OF THE MAJOR HISTOCOMPATIBILITY COMPLEX IN THE RESPONSE TO TUMORS IN RODENTS

A. Analysis of the Genetic Control of Immune Responses to Tumors or Tumor Induction

80. HISTOCOMPATIBILITY, IMMUNE RESPONSE, AND TUMOR INDUCTION: SPECIFIC IMMUNE RESPONSE GENES.

Benacerraf B, Green I, Bluestein HG, Ellman L
Harvard Med. Sch., Boston, Mass.
Transplant Proc; 3(3):1327-1331 1971

Two inbred guinea pig strains (2 and 13, developed by Wright) and Hartley random-bred lines were used to determine specific immune responsiveness. Three genes were identified in the guinea pig strains prior to experimentation: 1) the PLL gene, which controls responsiveness to poly-L-lysine, poly-L-arginine, copolymers of L-glutamic acid and L-lysine, and hapten conjugates of these polypeptides; 2) the GA gene, which controls responsiveness to L-glutamic acid and L-alanine; and 3) the GT gene, which controls responsiveness to a copolymer of L-glutamic acid and L-tyrosine. The first two genes are found in strain 2 but not in strain 13 in inbred animals; the GT gene is found in strain 13 but not in strain 2. In random-bred Hartley animals, the GT gene tends to segregate from PLL and GA responsiveness. All three strains were injected to determine if a fourth gene was necessary for responsiveness to bovine serum albumin (BSA) in low doses. Strain 2 exhibited linkage in the genes controlling responsiveness to PLL antigens, GA, and low doses of BAS; strain 13 did not respond. In random-bred animals, crossing over may have occurred between the PLL gene and the GA and BSA gene, since two responses were noted, one resembling strain 2 and the other resembling strain 13. Random-bred Hartley guinea pigs (78) were tested for the presence of a strain 2 specificity on their lymphocytes to determine if immune response genes are identical with genes controlling individual histocompatibilities or are very closely linked with them. Possession of the PLL gene was associated with susceptibility to lysis of the cells by antiserum of antiserum d; cells from PLL-negative animals were not susceptible to lysis by the antiserum. Responsiveness to antigens under control of specific immune responses genes is accompanied by antibody responses to haptens they bear, illustrating the gene-controlling carrier function. Conversely, it was discovered after further experiment that an antigen such as DNP-PLL can stimulate specific antibody synthesis in the absence of cellular immunity in animals lacking the PLL gene if it is administered bound to an immunogenic carrier.

81. INHIBITION OF CELL-MEDIATED CYTOLYSIS OF TRINITROPHENYL-DERIVATIZED TARGET CELLS BY ALLOANTISERA DIRECTED TO THE PRODUCTS OF THE K AND D LOCI OF THE H-2 COMPLEX (MEETING ABSTRACT).

Burakoff SJ, Germain RN, Benacerraf B

Dept of Pathology, Harvard Medical School, Boston, Ma 02115

Fed Proc; 35(3):826 1976

The role of the products of the K and D loci of the H-2 complex as target antigens for cytotoxic T cells generated to modified syngeneic cells has been investigated. We have confirmed the findings that normal spleen cells, when cultured with irradiated Tnp-derivatized syngeneic spleen cells, develop cytotoxic effectors that lyse most effectively a target cell that is: a) H-2 compatible with the stimulator; and b) Tnp derivatized. The cytolysis of these derivatized target cells is inhibited by: a) alloantisera directed to the products of the K and/or the D loci: and b) antihapten antibody, i.e., anti-Dnp antiserum. Effector cells generated to Tnp-derivatized stimulators also lysed, but less effectively, Tnp targets not sharing H-sD or H-2K products with the stimulator or effector cells; and this cross-reactive lysis occurred both for tumor and spleen targets. This cross-reactive lysis was also inhibited by antisera to the K and/or D loci of the Tnp-tumor or Tnp-spleen target cell. These data support the hypothesis that cytotoxic T cells in this model system are generated to new antigenic determinants which are determined by the modification of H-2 gene products. (Author Abstract)

82. THE ROLE OF GENETIC FACTORS IN THE COMBINED NEOPLASTIC EFFECTS OF VACCINIA VIRUS AND METHYLCHOLANTHRENE.

Duran-Reynals ML, Lilly F

Albert Einstein Coll. Med., Bronx, N.Y.

Transplant Proc; 3(3):1243-1246 1971

Studies with inbred mice of different strains revealed a close correlation in the strain distribution of skin susceptibility to vaccinia virus infection and susceptibility to tumorigenesis by methylcholanthrene (MC) painting. An inverse correlation was found between the skin response to vaccinia virus and MC painting, on the one hand, and the leukemogenic response to MC, on the other. Mice of the BALB/c strain, which are most susceptible to virus-induced skin ulceration and consequent papilloma formation, and which show a low incidence of spontaneous leukemia, were crossed with AKR mice which do not develop skin ulcers or tumors but show a high incidence of spontaneous leukemia. Experimental animals received one mg cortisone acetate sc for five days. Vaccinia virus was inoculated intradermally on the day of the last cortisone injection. Animals were painted once daily for five days with 1% MC beginning the day after virus inoculation. The F1 hybrids showed susceptibility to viral skin infection, but at a much lower level than the parental BALB/c strain. Susceptibility to papilloma production by MC painting was also of lower incidence than in the parental BALB/c strain. The F1 population, however, showed increased susceptibility to skin ulcer and tumor formation when treated by both vaccinia infection and MC painting (86% incidence). Only 4% of the F1 hybrids ultimately developed leukemia. In 78% of (BALB/c x AKR) x AKR backcross progeny treated with cortisone, vaccinia and MC showed skin sensitivity similar to the F1 hybrids, thus suggesting that skin susceptibility to vaccinia is determined by two independent genes. Fifty-six out of 107 of the virus-susceptible mice from the F1 backcross population developed skin tumors, compared to only two out of 31 virus-resistant mice. About half of the tumors which developed in the virus-susceptible mice progressed to malignancy. These results suggested that MC-induced skin tumorigenesis is determined by one gene which may be independent from those governing skin susceptibility to vaccinia. Results from this F1 x AKR backcross also showed a correlation between H-2 antigen and the occurrence of skin tumors; this correlation was most evident with the occurrence of malignant tumors. These results compared with those from BALB/c mice indicated that H-2 locus significantly suppresses skin susceptibility to MC-induced tumors. Study of leukemogenesis with respect to H-2 type, vaccinia susceptibility and skin tumors incidence showed that susceptibility to leukemogenesis and to skin tumor formation are mutually exclusive. A subsequent experiment indicated that the presence of Gross virus infection could inhibit tumorigenesis in MC-treated skin and could delay the onset of leukemia. Since the H-2 locus had previously been associated with susceptibility to leukemia in Gross virus-infected cells, it was considered possible that the apparent interference of natural infection by a leukemogenic virus with skin susceptibility to chemically induced tumors might depend significantly on H-2 type.

83. COMBINED NEOPLASTIC EFFECTS OF VACCINIA VIRUS AND 3-METHYLCHOLANTHRENE. II. GENETIC FACTORS.

Lilly F, Duran-Reynals ML

Albert Einstein Coll. Med., Bronx, N.Y.

J Natl Cancer Inst; 48(1):105-112 1972

Female BALB/c and AKR mice, female offspring of crosses of these two strains, and female offspring of the backcrosses (BALB/c x AKR)F1 x BALB/c and (BALB/c x AKR)F1 x AKR, were treated with combinations of cortisone (1 mg daily for five days sc), vaccinia virus id, and 3-methylcholanthrene (MCA) (1% solution painted daily for five days on shaved flank), and susceptibility to vaccinia dermal infection after cortisone, and to papillomas induced by MCA, was observed. BALB/c parents were highly susceptible to vaccinia dermal infection following cortisone, and to MCA tumorigenesis. AKR parents were resistant to both treatments. F1 offspring of (BALB/c x AKR) and (AKR x BALB/c) crosses were uniformly susceptible to vaccinia dermal infection, although these offspring were not as highly susceptible as the BALB/c parents. MCA tumorigenesis was enhanced in F1 hybrids by active vaccinia dermal infection; 15 of 31 F1 mice given cortisone and MCA, but no vaccinia, developed papillomas, whereas 37 of 43 mice given cortisone and MCA plus vaccinia developed papillomas. Although AKR parents were resistant to vaccinia, 78% of the offspring of the backcross (BALB/c x AKR)F1 x AKR showed an acute, ulcerative response to the virus. Statistical analysis indicated that susceptibility to vaccinia infection was due to two genes. Backcross studies also showed that enhancement of MCA tumorigenesis by vaccinia depended on mice being genetically susceptible to active infection with vaccinia. The incidence of spontaneous leukemia among mice of the (BALB/c x AKR)F1 x AKR generation was 41%. Leukemia was more frequent among backcross mice resistant to vaccinia infection than among those susceptible to vaccinia. (55 vs. 36%, resp) It is thought that H-2 or a closely linked gene is a factor in determining degree of susceptibility to MCA skin tumorigenesis in backcross offspring.

84. H-2 COMPATIBILITY REQUIREMENT FOR VIRUS-SPECIFIC T-CELL-MEDIATED CYTOLYSIS. THE H-2K STRUCTURE INVOLVED IS CODED BY A SINGLE CISTRON DEFINED BY H-2KB MUTANT MICE.

Zinkernagel RM

Scripps Clinic and Res. Foundation, La Jolla, CA 92037

J Exp Med; 143(2):437-443 1976

The genetic requirements for the lytic interaction of virus-specific cytotoxic T cells and target cells were studied in H-

2Kb mutant mice. Lymphocytic choriomeningitis or vaccinia virus-immune spleen cells of mice carrying a point mutation in the K region (B6 H-2ba, B6 H-2bf) could not lyse infected wild-type H-2Kb targets and vice versa. Yet, cytotoxic T cells specific for infected H-2Kba or H-2Kbf targets were generated during virus infections as shown by cold target competition experiments. The critical structure for the apparent restriction by the K or D regions of the H-2 gene complex of cytolytic interactions between T cells and virus-infected target cells are therefore each coded, at least as shown for the K region, by a single cistron. This finding is most readily accommodated within the altered self concept (postulating that T cells are specific for virus-modified self structures) but cannot exclude the possibility of a physiological interaction mechanism being responsible for the apparent H-2 restriction of virus-specific cytotoxic T cells.

85. ANTIBODY TO MURINE LEUKEMIA VIRUS: GENETIC CONTROL LINKED TO THE H-2 LOCUS IN PL MICE.

Nowinski RC, Doyle T
Fred Hutchinson Cancer Res. Center, 1124 Columbia St., Seattle, WA 98104
J Immunol; 117(1):350-351 1976

Immune nonresponsiveness in mice of the highly leukemic PL strain to endogenous ecotropic murine leukemia viruses (MuLV) is described. PL and C57L mice were used to breed F1 hybrid and backcross mice. The former strain has a high incidence of spontaneous leukemia, produces high titers of infectious ecotropic MuLV, and has the H-2u genotype. The latter strain has a low incidence of spontaneous leukemia, does not produce infectious ecotropic MuLV, and has the H-2b genotype. Radioimmune precipitation assays demonstrated that PL and C57L mice contained low titers of antibody against AKR MuLV; the hybrid (PL X C57L) produced antibody at a level higher than that of either parental strain. It was suggested that the PL genome contributed the immunogenic virus to the hybrid and that the C57L genome contributed a dominant immune response gene(s). To test these theories, a genetic backcross PL X (PL X C57L)F1 and (PL X C57L)F1 X PL was prepared. A comparison between the production of antibody to MuLV and the H-2 types showed a strong concordance in the segregation of these two traits. Humoral immune response to endogenous ecotropic MuLV in PL and C57L mice was controlled by a gene that mapped in the IXth linkage group. The PL strain had an immune nonresponsive allele of the gene, and the C57L strain had an immune responsive allele.

86. GENETIC BASIS OF SUSCEPTIBILITY TO VIRAL LEUKAEMOGENESIS.

Lilly F, Boyse EA, Old LJ
Sloan- Kettering Inst. Cancer Res., New York, N. Y.
Lancet; 2:1207-1209 1964

C3Hf/Bl mice, of blood group H-2 (histocompatibility-2) kk, when inoc at age 3-5 days with Passage A Gross virus, all developed leukemia within 87 days. After similar inoc of a group of C57BL mice (H-2 bb), only 1% developed leukemia in 87 days, 26% in 304 days. Among their reciprocal F1 hybrids (H-2 kb), less than 10% had become leukemic 320 days after inoc with Passage A virus. In backcross and F2 generations typed for H-2 by hemagglutination, homozygous kk mice were found to be highly susceptible to Gross virus-induced leukemia. Mice lacking k (bb) or heterozygous for k (kb) were much more resistant. H-2 type, as a single gene or a group of closely linked genes, is a major determinant of susceptibility to Gross virus leukemia. The association between histocompatibility factor and susceptibility to leukemia in the mouse suggests a possible similar association in man.

87. THE INFLUENCE OF H-2 TYPE ON GROSS VIRUS LEUKEMOGENESIS IN MICE.

Lilly F
Einstein Coll. Med., Bronx, N.Y.
Transplant Proc; 3(3):1239-1242 1971

Susceptibility and resistance to Gross virus leukemogenesis was found to be associated with the H-2 locus in mice chromosomes. Further analysis showed this locus to have at least two genes associated with it. One of these, called Rgv-1, appeared to be closely linked to the H-2 region; the existence of the other gene, however, has yet to be decisively confirmed. The authors attempted to find another gene locus by crossing two inbred mice strains, B10.BR and C3H-OH, both of which were highly susceptible to Gross virus leukemogenesis when inoculated three to five days after birth. When these homozygous non-resistant mice were crossed with each other, it was discovered that the F1 generation was no longer susceptible to the virus. Thus, the recessive genes for the trait in the parents are not allelic, but rather reside at different loci in the genome. When the F2 generation was mathematically analyzed for leukemogenesis phenomena, taking "background" from the F1 generation into account, it was found that the number of genes involved was probably either two or four. A Mendelian genetic model was set up to prove this. Four genotypes were proposed, the phenotypes of which were resistant and susceptible at a ratio of 9:7. Data obtained from the experiment yielded 54% susceptible (105 mice of 194). It appears that genes other than H-2 might determine the success or failure of the virus in infecting host cells, whereupon H-2 type determined whether or not the infection, once established, will lead to the appearance of leukemia and to death. It is concluded that H-2 does not appear as an all-or-none determinant of leukemia susceptibility.

88. IMMUNOLOGICAL AND GENETIC FACTORS IN MURINE VIRUS-INDUCED LEUKEMIA.

Chieco-Bianchi L, Collavo D, Pennelli N, Tridente G
Div. Cancer Res., U. Padua, Italy
Bibl Haematol; 36:234-239 1970

One group of 1- to 3-day-old mice of C3Hf/Gs (H-2k), C57BL(H-2b) and (C3Hf/Gs x C57BL)F1 strains were injected sc with 0.05 ml of 20% cell-free extract from C3Hf/Gs mice with passage A Gross virus-induced primary leukemia; a second group of adult C3Hf/Gs and F1 hybrids, both neonatally virus-injected and normal, were given ip repeated injections of dilute cell-free leukemic extract containing passage A Gross virus for the preparation of sera against target C3Hf/Gs passage A leukemic cells. Results showed that while serum from C3Hf/Gs mice that were neonatally virus-injected and immunized when adults contained practically no antibodies, the serum from C3Hf/Gs mice receiving no virus neonatally but immunized when adults showed significant antibody titers. Sera taken from 2 F1 hybrid groups exhibited a remarkably similar cytotoxic activity regardless of the fact that one group had been treated with virus neonatally and the other group remained untreated. All these sera were devoid of detectable cytotoxicity when tested on normal C3Hf/Gs cells as were sera from normal C3Hf/Gs and F1 hybrid donors when tested on leukemic C3Hf/Gs cells. The oncogenic effect of passage A group virus was neutralized by sera from both C3Hf/Gs and F1 hybrid mice which had received virus when adults; serum from F1 hybrids neonatally virus-injected and immunized when adults was equally effective and showed a remarkable decrease in leukemia incidence in the test animals; serum from C3Hf/Gs mice, neonatally injected with virus and subsequently immunized did not neutralize the oncogenicity of the virus.

89. THE ROLE OF GENETICS IN GROSS VIRUS LEUKEMOGENESIS.

Lilly F
Albert Einstein Coll. Med., Bronx, N. Y.
Bibl Haematol; 36:213-220 1970

Hybrid (BALB x C57BL) x BALB backcross generation mice and mice of the (B10.BR x C57BL) x N10.BR backcross generation inoculated with Gross virus and typed for the histocompatibility-2 locus of the 9th chromosome were observed for leukemia incidence. Heterozygous H-2b/H-2d mice among the first hybrids showed a lower incidence of leukemia among 2 groups with values of 3% and 37% compared to their homozygous H-2d/H-2d littermates with values ot 44% and 76%. Among the second hybrids the H-2k/H-2k segregants showed a leukemia incidence that was consistently higher and occurred sooner than that among heterozygous H-2k/H-2b segregants; however, the difference between the 2 classes of mice appeared greater when virus preparations of lower potency were used than when high-titer preparations were used. The incidence of leukemia induced by Gross virus among mice of various strains indicated a significantly greater susceptibility to leukemogenesis among BALB/c strain mice than mice of the A strain and presented a more complex association between the histocompatibility locus and leukemogenesis.

90. INHERITANCE OF SUSCEPTIBILITY TO FRIEND MOUSE LEUKEMIA VIRUS. VI. RECIPROCAL ALTERATION OF INNATE RESISTANCE OR SUSCEPTIBILITY BY BONE MARROW TRANSPLANTATION BETWEEN CONGENIC STRAINS.

Odaka T, Matsukura M
Inst. Med. Sci., Takanawa, Tokyo
J Virol; 4(6):837-843 1969

A study of the effects of bone-marrow transplantation in 2 newly established mouse strains which are congenic with the DDD and C57BL strains suggest that the effect of the gene locus (Fv) responsible for sensitivity or resistance to Friend leukemia virus (FLV) is expressed in bone-marrow derived cells and that they play a major role in the development of splenomegaly which occurs with FLV infection. Congenic strains of mice have the same major histocompatibility gene but have different Fv loci. Splenomegaly did not develop in mice with the Fvr/Fvr genotype (DDD-Fvr, C57BL/6) while mice with the Fvs/Fvs genotype (DDD, C57BL/6-Fvs) developed splenomegaly even when they were inj ip with 10-3-10-5-fold the dose given resistant strains. Mice of each strain were exposed to 700-900 r of whole-body irradiation, inoc with bone marrow cells from either syngenic or congenic mice, and challenged later with FLV. Leukemia did not develop when Fvs/Fvs mice were given bone marrow transplants taken from Fvr/Fvr mice, but Fvr/Fvr mice developed marked splenomegaly after receiving Fvs/Fvs bone marrow cells. High titers of infectious virus were found in the enlarged spleens of C57BL/6 mice which are ordinarily resistant to FLV infection. These changes in the response of mice to FLV infection appear to be caused by repopulation of destroyed tissues by the transplanted bone marrow cells.

91. GENETIC CONTROL OF MURINE VIRAL LEUKEMOGENESIS.

Lilly F, Pincus T
Albert Einstein Coll Med., Bronx, N. Y.
Adv Cancer Res; 17:231-277 1973

The genetic control of murine viral leukemogenesis is reviewed. Early genetic studies of murine leukemia are discussed with regard to the high leukemia mouse strains, leukemogenesis in hybrids of high and low leukemic strains, and leukemia induced by various chemical and physical agents. The genetic control of Friend virus susceptibility is considered in terms of the host range of Friend virus, the genetic basis of the host range patterns, congenic strains for Friend virus susceptibility, the basis of focus formation by Friend virus, and other genes affecting Friend virus susceptibility. The genetic control of naturally occurring leukemia viruses is also discussed with attention to the susceptibility to leukemogenesis by naturally occurring viruses, tissue culture studies of naturally occurring leukemia viruses, and the genetic basis of N- and B-tropism of the Fv-1 gene. The H-2-linked genetic control of Gross virus and Friend virus leukemogenesis is also considered, as are genes affecting spontaneous and chemically induced leukemogenesis and genes determining leukemia virus-associated antigens.

92. GENES OTHER THAN H-2 DETERMINING SUSCEPTIBILITY TO FRIEND VIRUS.

Steeves RA
Roswell Park Mem. Inst., Buffalo, N.Y.
Transplant Proc; 3(3):1237-1238 1971

C57BL mice are known to be resistant to Friend virus-induced splenomegaly and spleen focus formation. Recent studies have shown that genes at two unlinked loci, called Fv-1 and Fv-2, are mainly responsible for this resistance. It was found that the as yet unmapped Fv-1 gene seems to control the relative susceptibility of mice to all murine leukemia viruses. The Fv-1b allele conferred reciprocal susceptibility and resistance. It is postulated that the FV-1b gene may inhibit an N-tropic helper virus which is found in a high titer as a lymphatic leukemia virus in stocks of Friend virus complex.

93. A MAJOR GENETIC LOCUS AFFECTING RESISTANCE TO INFECTION WITH MURINE LEUKEMIA VIRUSES: I. TISSUE CULTURE STUDIES OF NATURALLY OCCURRING VIRUSES.

Pincus T, Hartley JW, Rowe WP
Natl. Inst. Allergy. Infec. Dis., Natl. Inst. Hlth., Bethesda, Md.
J Exp Med; 133(6):1219-1233 1971

The host range of N- and B-tropic murine leukemia viruses was investigated by inoculating mouse embryo cell cultures prepared from mice of various strains with the virus sub-type; "N-tropic" viruses initiated infection 30-1000 times more efficiently on NIH Swiss embryo cells than on BALB/c cells; on the other hand, "B-tropic" viruses infected BALB/c cells more efficiently than NIH Swiss cells. Embryo cells from 16 mouse strains were inoculated either with N-tropic virus (prepared from AKR-L1 cells), or with B-tropic virus (prepared from BALB/c-S2B cells), or with a Moloney virus which infected both BALB/c and NIH Swiss cells with equal efficiency (i.e, "NB-tropic" virus). All 16 mouse cells resembled either NIH Swiss mouse or BALB/c mouse cells in showing at least 60-fold greater sensitivity to either an N-tropic or a B-tropic virus. The classification of strains of mouse cells as sensitive to N-tropic or to B-tropic virus (N-type or B-type cells) was not related to the H-2 allele make-up of the cells; the H-2b, H-2d, and H-2k alleles were represented in both N-type and B-type mouse cells. When embryo cells from F1 hybrid offspring of N-type and B-type parent mice were inoculated with N-tropic or B-tropic virus, it was found that the offspring were 100-fold less sensitive to either virus than parent mice; this suggested that resistance to virus was genetically dominant in mice. The F1 offspring of B-type-N-type and of N-type-B-type mouse matings were backcrossed to parental N- and B-tropic strains, and cultures from backcross embryos were inoculated with N- and B-tropic virus. Backcross embryo cells showed no segregation for sensitivity to N- or B-tropic virus; this finding suggested that a single genetic locus determines in vitro susceptibility of mouse cells to murine leukemia viruses.

94. HISTOCOMPATIBILITY GENES (THE H-2 COMPLEX) AND SUSCEPTIBILITY TO MAMMARY TUMOR VIRUS IN MICE.

Muhlbock O, Dux A
Netherlands Cancer Inst., Amsterdam
J Natl Cancer Inst; 53(4):993-996 1974

Congenic strains of C57BL/10ScSn (H-2b) mice with the haplotypes H-2a, H-2d, H-2f, H-2i, H-2k, H-2m, H-2r, and H-2pa received mammary tumor virus (MTV) by foster-nursing on (C3H x 020)F1 females. After force breeding of mice, the mammary tumor incidence showed remarkable differences among strains. The strains with the haplotypes H-2b and H-2m were relatively resistant, while those with H-2f, H-2i, H-2d, and H-2a were highly susceptible. The others showed intermediate susceptibility. Comparison of the results, especially with the recombinant strains, indicated that genes in or near the D end of the H-2 locus determined the susceptibility to MTV: the subloci 2Db and 2Dq determined resistance, and 2Dd and 2Df determined high susceptibility.

95. HOST-GENE CONTROL OF TYPE-C RNA TUMOR VIRUS EXPRESSION AND TUMORIGENESIS IN INBRED MICE (MEETING ABSTRACT).

Meier H, Taylor BA, Cherry M, Huebner RJ
Jackson Lab., Bar Harbor, Me.
Proc Natl Acad Sci USA; 70(5):1450-1455 1973

Genetic hybridization experiments involving a high leukemia incidence mouse strain (ADR/J) and a low incidence strain (C57L/J), their reciprocal F1 hybrids, and the F2 and backcross generations of this cross were conducted to determine whether or not a relationship exists between endogenous viral expression and tumorigenesis. A highly significant (P less than 0.001), predictable association was found between the expression of complete infectious virus, or gs antigen, as determined by complement fixation or the COMul test, and tumorigenesis in later life. Most tumors were thymic leukemia or reticulum cell sarcoma (RCA). Of 124 tumors occurring in parental C57L/J mice, 110 developed a gs-positive animals. All gs- and virus-positive AKR/J mice and all but one of the F1 hybrids developed tumors. All 56 gs-positive F2 progeny developed tumors. It was thus concluded that the endogenous C-type RNA viral genome was a major determinant of at least leukemias and RCS. In addition, a possible minor effect of the H-2 histocompatibility locus on leukemogenesis was found in F2 mice.

96. HOST CONTROL OF ENDOGENOUS MURINE LEUKEMIA VIRUS GENE EXPRESSION: CONCENTRATIONS OF VIRAL PROTEINS IN HIGH AND LOW LEUKEMIA MOUSE STRAINS (MEETING ABSTRACT).

Strand M, Lilly F, August JT
Albert Einstein Coll. Med., Bronx, N. Y.
Proc Natl Acad Sci USA; 71(9):3682-3686 1974

Two of the major molecular components of murine leukemia virus particles, the internal protein (p30) and the envelope glycoprotein (gp69/71) were measured in the spleens of normal, 6-10, week old mice of various inbred strains and crosses. Radioimmunoassay measurement of the viral p30 and gp69/71 proteins was carried out by a heterologous assay system with anti-feline virus serum and murine virus protein. The reaction mixtures contained the following: 5 microliter normal rabbit serum, 10 microliter of purified virus or viral proteins as competing proteins, and 10 microliter of rabbit antiserum to Theilen feline leukemia virus at a concentration that precipitated approximately 50% of the 125I-labeled antigen. The reaction mixture was incubated at 37 C for 15-18 hr, 0.04 ml of horse or goat anti-serum to rabbit IgC added, incubation continued for an additional two hr followed by overnight incubation at 2-4 C. The 125I-labeled antigen present in the precipitate after centrifugation was measured in a gamma counter. Both proteins were detected in virtually all mice. Extracts of high leukemia, high murine leukemia virus strains (AKR, C58, PL) showed high levels of both proteins; extracts of other strains usually showed lower levels. Very little gp69/71, however, could be detected in spleens of BALB mice, and this trait was dominant in crosses with AKR and DBA/2, both of which express a high level of gp69/71. Thymus-deficient BALB/c-nu/nu mice, showed a higher concentration of gp69/71 typical of other low leukemia strains, suggesting that the virtual absence of the protein in normal BALB/c mice may result from immunologic suppression. With C57L, C57BL/10Sn, and C57BL/6 strains the concentration of p30 was lower, in some cases much lower, than would be expected from the concentration of gp69/71. DBA/2 mice showed high levels of gp69/71, ten-fold greater than that of p30, whereas congenic DBA/2-Fv-1b mice showed the opposite pattern. Mice of 129-GIX-strain showed no detectable levels of either p30 or gp69/71 proteins, although the congenic 129(GIX+) showed appreciable levels of both. The absence of these proteins in 129-GIX-mice is a recessive trait, as F1 hybrids of AKR and DBA/2 showed appropriate levels of both proteins. It is concluded that expression of viral p30 and gp69/71 proteins in mice is not coordinately linked and that expression is complex, being influenced by several genes, including Gv-1, H-2, Fv-1, and probably others.

97. LOCATION OF THE SECOND GENE REQUIRED FOR EXPRESSION OF THE LEUKEMIA-ASSOCIATED MOUSE ANTIGEN GIX.

Stockert EH, Sato H, Itakura K, Boyse EA, Old LJ, Hutton JJ
Sloan-Kettering Inst. Cancer Res., New York, N.Y.
Science; 178(4063):862-863 1972

The location of the Gv-2 gene locus was established in a three-point cross with Gpi-1 (glucose phosphate isomerase) and Hbb (beta chain of hemoglobin). Gv-2, along with Gv-1, controls GIX, a cell surface antigen, found on thymocytes of some (i.e, GIX+) mouse strains and not on others. Murine leukemia virus causes GIX antigen to be expressed on cells of genotypes which normally yield the GIX-phenotype, that is, on cells of mice lacking either the Gv-1+ allele or the Gv-2+ allele, or both. Gv-1 had been located on linkage group IX, 36 U from H-2. Gv-2 was located on linkage group I (chromosome 7), 33.6 plus or minus 5 U from Hbb, with the gene order: Gpi, Hbb, Gv-2.

98. RELATION OF CHROMOSOME 4 (LINKAGE GROUP VIII) TO MURINE LEUKEMIA VIRUS-ASSOCIATED ANTIGENS OF AKR MICE.

Ikeda H, Stockert E, Rowe WP, Boyce EA, Lilly F, Sato H, Jacobs S, Old LJ
Sloan-Kettering Inst. Cancer Res., New York, N. Y.
J Exp Med; 137(4):1103-1107 1973

The locations of genes coding for the expression of three murine leukemia virus (MuLV)-associated antigens - GIX (cell-surface antigen), GCSA (Gross cell-surface antigen), and gs (group specific viral antigen) - have been determined from results of segregating crosses of young AKR mice (an Ev-1a strain) with different Fv-1b mouse strains. The results indicate that the genes specifying or controlling the expression of all three antigens are located in linkage group (LG) VIII of chromosome 4 of the AKR mouse. The data strongly indicates that the locus specifying GIX is about 19 map units from the Fv-1 (Gpd-1) region. Whether the gs phenotype is an expression of virus titer regulated by Fv-1 or an independent Mendelian trait governed by a discrete locus in LG VIII could not be determined. The GIX and GCSA phenotypes of the AKR backcross segregants were invariably associated, indicating a close linkage between their controlling genes. The GIX-determining gene in LG VIII of AKR mice is apparently equivalent to Gv-1, which controls expression of the same antigen in strain 129 mice, but which in 129 mice is located in LG IX. Since the estimated distance of Gv-1 from the H-2 locus in 129 mice is considerable (37 units), the possibility of pseudolinkage cannot be excluded in this case.

99. GENETIC CONTROL OF THE GROUP-SPECIFIC ANTIGEN OF MURINE LEUKAEMIA VIRUS.

Taylor BA, Meier H, Huebner RJ
Jackson Lab., Bar Harbor, Maine
Nature New Biol; 241(110):184-185 1973

A second example is reported of single gene control of the expression of group-specific antigen (gs-AG) in inbred mice A number of congenic resistant (CR) lines were tested for their effects on gs-AG expression in the spleens of 5-7 wk old mice. Each tested line differed from their common inbred partner strain, C57BL/10Sn (B10), by a single histocompatibility gene. In contrast to B10 and two CR lines, B10.129(5M) and B10.129(21M), which were gs-AG negative, spleens of B10.D2(58N) mice (58N) reacted consistently gs-AG positive in a direct complement fixation test. Genetic analysis of the difference in gs-AG titers between B10 and 58N suggested that 58N contained a single contaminant gene from strain DBA/2J that controls gs-AG expression. This was confirmed by segregation data for F2 and backcross generations obtained by crossing the B10 and 58N strains. Joint segregation of gs-AG with beta-chain-hemoglobin locus (Hbb) revealed no significant deviation from free recombination in both the F2 and backcross generations, nor was any association with the p locus observed. In crossed strains, lack of gs-AG expression was dominant over expression and the presence of gs-AG was unaccompanied by detectable complete, replicating leukemia virus.

100. HISTOCOMPATIBILITY GENES AND SUSCEPTIBILITY TO MAMMARY TUMOR VIRUS (MTV) IN MICE.

Muhlbock O, Dux A
Netherlands Cancer Inst., Amsterdam
Transplant Proc; 3(3):1247-1250 1971

Mammary tumor virus (MTV) has three different variants. Two of these variants have been investigated in this work: C3H-MTV, which is transmitted by the mothers' milk, and GR-MTV, transmitted at conception by either the sperm or the ovum. Congenic resistant (CR) strains and normal strains of the C57BL/10 mice were subjected to four separate experiments. In the first, newborn mice were given C3H-MTV by way of the milk. It was found that the CR-strains in which the H-2b allele of the B10-strain mice is replaced by the alleles 2a, 2d, 2f, 2k and 2m have a significantly higher incidence of tumor formation than both the B-10 strain and the CR-strains with H-2b allele but with different weak histocompatibility loci 1am, 1b, 3b, 8b and 9b. In the second experiment 2-month-old females of the CR-strain were injected ip with cell-free mammary tumor extract. Mammary tumor incidence was found to be higher in the females with the H-2 alleles 2a, 2f and 2m than in females with the H-2b allele. Replacing the different alleles at the weak histocompatibility loci had no influence. In the third experiment, GR-MTV was transmitted by males or females at conception to hybrid F1 mice. Hybrids of all CR strains had a high percentage of tumors, regardless of histocompatibility loci. Lastly, GR-MTV was given to newborn females through the lactation process. Females carrying the H-2b allele were found to be slightly more susceptible to tumors than those carrying other alleles. The influence of the H-2 locus on the susceptibility to MTV is thus only apparent with C3H-MTV.

101. IMMUNE DEFICIENCY, AUTOIMMUNITY AND AGING.

Yunis EJ, Fernandes G, Greenberg LJ
Univ. Minnesota Medical Sch., Minneapolis, MN
Birth Defects; XI(1): 185-192; 1975

Aging in man and lower animals is accompanied by a decline in vigor of both humoral and cell-mediated immunity, the latter suffering a very sharp decline in man after age 50. Certain autoimmune phenomena also appear to be related to aging. Although the mechanism that controls the normal tolerance to self-antigens is not clear, pathological changes in the thymus have been linked to diseases such as myasthenia gravis and lupus erythematosus, which have autoimmune components. Aged mice from short-lived strains such as strain A show a propensity to produce autoantibodies and a decline in T lymphocytes. Both the survival and the immunological function of neonatally thymectomized strain A mice could be better improved either by ip thymus grafts or 10^8 spleen cells given ip from young syngeneic mice than by thymus grafts or spleen cells from aged mice. Survival at 8 mo was 85% and 35% and immunological reconstitution 73% and 11% with spleen cells from 4- and 24-mo sources, respectively; survival at 8 mo was 46% and 23% and immuno- logical reconstitution 32% and 5% with thymus grafts from 4- and 24-mo sources, respectively. The decline of postthymic cells or T cells during aging is attributable to a thymic defect or to involution of the thymus itself; the stem cell compartment in aged mice is not abnormal. Susceptibility to a variety of murine autoimmune and neoplastic diseases is partially under the control of a gene (or genes) that is closely associated with the region of the H-2 gene complex.

102. GENETIC CONTROL OF ONCOGENESIS BY MURINE SARCOMA VIRUS MOLONEY PSEUDOTYPE. I. GENETICS OF RESISTANCE IN AKR MICE.

Colombatti A, Collavo D, Biasi G, Chieco-Bianchi L
Inst. of Pathological Anatomy, Univ. of Padova, Italy
Int J Cancer; 16(3):427-434 1975

The differential susceptibility to M-MSV in hybrids of AKR mice was studied. Injection (im, thigh) of Moloney mouse sarcoma virus (M-MSV) in adult mice of several inbred strains revealed that all strains except AKR are highly susceptible (80-100%) to M-MSV tumor development. F1 hybrids between AKR and CBA, DBA/2 or NIH mice are as resistant (93%) as the parental AKR strain, which indicates that resistance is transmitted as a dominant character. First backcross mice to the susceptible parent show a 3:1 ratio of resistant to susceptible mice. This is the expected ratio for two segregating loci which independently confer resistance. The incidence of resistant F2 mice is somewhat lower than expected. Further support for the two-gene hypothesis was obtained in second backcross mice. The major genes affecting murine leukemia virus infection (Fv-2 and H-2) do not seem to play any role in this system and no linkage was found with Thy.1 and albino, dilute, agouti and brown markers.

103. GENETIC CONTROL OF ONCOGENESIS BY MURINE SARCOMA VIRUS MOLONEY PSEUDOTYPE. II. A DOMINANT EPISTATIC SUSCEPTIBILITY GENE.

Colombatti A, Collavo D, Biasi G, Chieco-Bianchi L
Inst. of Pathological Anatomy, Univ. of Padova, Italy
Int J Cancer; 16(3):435-441 1975

The tumor response pattern of F1, F2, and backcross hybrids between resistant AKR and susceptible strains (C57Bl/6, BALB/c, and B10BR) of mice following injection with the Moloney mouse sarcoma virus (M-MSV) was studied. Fv-1 typing was determined by progeny testing using the Friend virus spleen focus assay, and a humoral cytotoxic assay on peripheral leukocytes was performed for the detection of

H-2K haplotype. Among adult hybrids between AKR and B6, BALB, or B10BR, 86% developed tumors within ten days after M-MSV injection; this was nearly the same percentage as was observed in the susceptible parents. Segregation was observed in the first backcross (Bc1) and F2 mice, and the segregation ratios up to Bc3 mice fit a one-gene model. In tests in which susceptible F1 hybrids were mated with a third nonparental strain, 48% of the progeny were susceptible, indicating that in some strains and their hybrids a dominant epistatic gene was masking the resistant phenotype. Progeny testing for Fv-1 showed no close linkage between susceptibility and H-2, although a slight suggestion of linkage with albino phenotype was found in F2, but not Bc1, mice. The data conclusively indicate that M-MSV tumor induction is under host genetic control. At least three genes appear to govern the tumor response in the offspring of crosses with AKR mice: two dominant autosomal loci independently confer resistance to M-MSV tumor development in AKR; and a dominant autosomal epistatic gene masks the resistant phenotype in some individuals. There is also some evidence that additional factors may exist which exert a minimal influence on tumor response.

104. LYMPHOMA INDUCED BY HERPES VIRUS: RESISTANCE ASSOCIATED WITH A MAJOR HISTOCOMPATIBILITY GENE (MEETING ABSTRACT).

Longenecker BM, Pazderka F, Gavora JS, Ruth RF
Departments of Immunology and Zoology, University of Alberta,Edmonton, Alberta and Agriculture Canada, Ottawa, Ontario
Fed Proc; 35(3):827 1976

Marek's disease (MD) is a herpes virus (MDV) induced lymphoma of chickens preventable by vaccination and genetic resistance. The B-locus is the major histocompatibility locus of chickens. Line-N which was selected for resistance to MD is uniformly homozygous for the B^{21} allele. Line-7 which was selected for susceptibility to MD is uniformly homozygous for the B^2 allele. Line-7 was crossed with line-N to produce F1's which were crossed to produce F2's (B^2/B^2, B^2/B^{21}, B^{21}/B^{21}). F2's were exposed to MDV at hatching. The rate of loss of B^2/B^2 chicks due to MD exceeded the loss of B^2/B^{21} chicks which exceeded the loss of B^{21}/B^{21} chicks. Furthermore, we have detected B^{21} in several unrelated lines of chickens which are survivors of severe outbreaks of MD and in the Red Jungle Fowl, the progenitor of the species. Our results suggest a functional and ancient, association of a major histocompatibility antigen with relative insusceptibility to lymphoma induced by herpes virus. (Author Abstract)

105. DIFFERENTIAL BEHAVIOUR OF TRANSPLANTED MOUSE LYMPHOMA LINES IN GENETICALLY COMPATIBLE HOMOZYGOUS AND F1 HYBRID MICE.

Hellstrom KE
Inst. Tumor Biol., Karolinska Inst. Med. Sch., Stockholm
Nature (Lond); 199:614-615 1963

Variant sublines of a lymphoma LNSF originally from an A/Sn x A.SW F1 hybrid (2 isogenic resistant mouse strains differing at the histocompatibility-2 locus) were obtained with irreversible loss of the histocompatibility-2 antigens specific for A (4 variants), and for A.SW (2 variants). On comparison of the variants with the unselected F1 tumor from which they were derived for ability to grow in mice of the original F1 genotype it was found that the variants grew less well in the F1 hybrids than the unselected lines. Mechanism of the phenomenon remains to be clarified, although two alternatives appear possible, one based on a graft-versus-host reaction, the other on a host-versus-graft response. The results also suggest that cells with certain antigens may be selected from large populations of cells lacking these antigens, and experiments of this type are in progress.

106. RELATIONSHIP OF POLYDACTYLISM, TUMOR SUSCEPTIBILITY AND THE H-2 COMPLEX OF THE STRONG'S POLYDACTYLOUS DESCENTS IN MICE (MEETING ABSTRACT).

Johnson F, Flounders M, Matsunaga H, Strong LC
Biol. Station Roswell Park Mem. Inst., Springville, N.Y.
Proc Am Assoc Cancer Res; 4(1):32 1963

A relationship was found between the absence of polydactylia and susceptibility to a transplantable anaplastic carcinoma in Strong mice and their polydactylous descents. Genetic analysis suggests relation or linkage to the H-2 complex locus, with the pBr/St strain having the genotype H-2x H-2x. The incidence (percentage) of (1) polydactylism and (2) tumor susceptibility in the pBr/St mouse was 0 and 97.4; polydactylous sublines (poly 1, poly 2, poly 3, poly 4, and poly 5) results were, resp: 80 and 10.7, 4 and 46.6, 60 and 0, 60 and 0, and 0.4 and 73.2.

107. INCIDENCE OF BENZOPYRENE-INDUCED TUMORS IN LINES OF MICE GENETICALLY SELECTED FOR "HIGH" AND "LOW" ANTIBODY PRODUCTION.

Biozzi G, Stiffel C, Mouton D, Bouthillier Y, Decreusefond C
Broussais Hosp., Paris, France
Transplant Proc; 3(3):1333-1336 1971

Random-bred Swiss mice were genetically selected for "amount of antibody" produced after immunization with optimal doses of heterologous erythrocytes. The mice were progressively separated into "high" and "low" responding lines during selective breeding until a maximal interline separation was reached after 20 generations. The two lines were first tested by spleen inoculation for humoral antibody responsiveness to histocompatibility antigens. It was found that the "high" line produced a high titer of cytotoxic antibodies to "low" line spleen cells, while the "low" line produced a low titer in response to "high" line spleen cells; however, interline skin graft studies showed rejection in both strains at the same time. Genetic selection operated only on humoral responsiveness, while cell-mediated immunity was not affected. Finally, mice of the sixteenth and nineteenth generations of both the "high" and the "low" lines were injected with benzopyrene. Tumors developed much more readily and grew more rapidly in the "low" line, showing that resistance to tumor formation is probably related to humoral immune responsiveness.

108. THE GENETIC CONTROL OF T CELL- MEDIATED IMMUNITY AGAINST THE DBA/2 MASTOCYTOMA P-815. II. LOW RESPONSIVENESS IN T CELL-MEDIATED CYTOTOXICITY ACCOMPANIED BY THE INABILITY TO PRODUCE ANTIBODIES IN A SECONDARY RESPONSE.

Kolsch E
Heinrich-Pette Inst. Exp. Virol. Immunol., Univ. Hamburg, Hamburg, W. Germany
Eur J Immunol; 5(8):527-532 1975

A study of the low responsiveness in T-cell mediated cytotoxicity of DBA/2 mouse P-815 mastocytoma (MC) in DBA/1 mice is reported. When DBA/1 and C57BL/6 mice were treated with cyclophosphamide (CP, 4 mg s.c) 3 d before immunization with P-815 cells (2×10^6 to 1×10^7 cells i.p), it was shown that T-cell cytotoxicity was enhanced by CP in the high responder C57BL/6, but was not restored by CP in the DBA/1, suggesting that their low responsiveness is not due to feedback inhibition through antibodies. When both strains of mice were challenged with 5×10^6 P-815 cells, cytotoxic antibodies were detected in C57BL/6 but not in DBA/1. This same result was confirmed in the more sensitive cytotoxicity inhibition assay (CIA), and CIA demonstrated further that optimal antibody responses are reached in C57BL/6 after priming with 5 x 10^6 P-815 cells, resulting in serum immune capacity 20-30 x higher than that of equally primed DBA/1. It was consistently found that the inhibitory activity on H-2d effector cells of DBA/1 sera 1 wk after primary immunization was 2-4 x higher than that of C57BL/6 despite the fact that normal sera from unprimed mice of the 2 strains do not differ in their anti-H-2d background activity. Living allogeneic spleen cells were required for enhancement of antibody production against P-815 cells in DBA/1 mice. (25 refs)

B. Immune Reactivity to Tumor Antigens and Major Histocompatibility Products

109. T CELL RESPONSE IN H-2 CONGENIC MICE TO FRIEND VIRUS-INDUCED TUMOR CELL LINES (MEETING ABSTRACT).

Blank KJ, Freedman HA, Lilly F
Albert Einstein College of Medicine, Bronx, N.Y. 10461
Proc Am Assoc Cancer Res; 17:96 1976

Mice immunized with cells from a syngeneic Friend virus (FV)-induced cultured tumor cell line produced T killer cells which did not kill FV-induced tumor cells derived from mice differing genetically only at the H-2 region. Congenic BALB.B (H-2**b) and BALB.K (H-2**k) mice histocompatible tumor cells from cell lines (HFL/b from BALB.B and HFL/k from BALB.K) induced in vivo by FV. Tumor cells from these lines express the FMR cell surface antigen and the gp70 virus envelope antigens. In an in vitro lymphocyte mediated cytotoxicity (LMC) assay, peritoneal exudate cells (PEC) from BALB.K and (BALB.B x BALB.K) greater than F1 mice immunized with HFL/k killed both HFL/k and HFL/bk (induced in (BALB.B x BALB.K) greater than F1 mice) but not HFL/b whereas PEC from BALB.B and (BALB.B x BALB.K) greater than F1 mice immunized with HFL/b killed both HFL/b and HFL/bk but not HFL/k. Pretreatment of the PEC with anti-thy 1 and C' abolished killing in the LMC assay. Thus, the tumor cells used for immunization and as target cells must share a common H-2 haplotype for T cell killing to occur. It appears that T lymphocytes do not respond to virus-induced antigens per se since HFL/b, HFL/k and HFL/bk all express the same vital antigens but perhaps to some virus-induced alteration in the H-2 molecule. (Author Abstract)

110. STUDIES ON THE ROLE OF THE HOST IMMUNE RESPONSE IN RECOVERY FROM FRIEND VIRUS LEUKEMIA: I. ANTIVIRAL AND ANTILEUKEMIA CELL ANTIBODIES.

Chesebro B, Wehrly K
Natl. Inst. Allergy and Infectious Diseases, Rocky Mountain Lab., Hamilton, Mont. 59840
J Exp Med; 143(1):73-84 1976

The humoral immune response to Friend virus (FV) leukemia was studied in congenic F1 mice differing in their incidence of recovery from leukemia. Anti-FV serum, containing FV neutralizing antibodies as well as cytotoxic antibodies for FV-induced cell surface antigens, was given to (B10.A x A.BY)F1 mice at 1, 3, or 6 days after FV inoculation, and mice were examined for spleen foci on the 9th day. Anti-FV serum given 1 or 3 days after virus reduced the number of virus-induced spleen foci. Antiserum given on day 6 was not effective. Anti-viral neutralizing antibodies rose in titer in vivo concurrently with disappearance of viremia and fall in spleen virus levels. Cytotoxic antileukemia cells antibodies also appeared at this time. Passive transfer of these antibodies could inactivate low numbers of leukemia cells in vivo; however, mice of both high and low recovery genotypes produced antibodies in equal titer and recovered from viral infection in spite of striking differences in recovery from leukemic splenomegaly. Female (B10.A x A.BY)F1 and C57BL/10 x A.BY)F1 mice were inoculated iv with 15,000 focus forming units of B-tropic FV, and were subsequently examined for viremia and antibodies. Mice lacking C57BL genes did not produce antibodies or recover from viremia except in rare instances. Recovery from splenomegaly was found to be influenced by 3 or more C57BL genes independent of the H-2 complex. The data suggest that neutralizing anti-FV antibody may play a major role in elimination of viral infection, and that this may be a necessary, but not alone sufficient, step in the process of recovery from FV-induced splenomegaly.

111. CORRELATION OF EARLY MURINE LEUKEMIA VIRUS TITER AND H-2 TYPE WITH SPONTANEOUS LEUKEMIA IN MICE OF THE BALB/C X AKR CROSS: A GENETIC ANALYSIS.

Lilly F, Duran-Reynals ML, Rowe WP
Albert Einstein Coll. Med., Bronx, N.Y.
J Exp Med; 141(4):882-889 1975

The relationship between the expression of endogenous murine leukemia virus (MuLV) and spontaneous leukemia in a segregating population of mice was investigated. All of the mice possessed two or more copies of the Akv genes, which had previously been shown to be a determinant of MuLV expression, but varied genotypically in respect to regulatory genes capable of interfering with the expression of the disease. MuLV testing and H-2 typing were carried out on 335 (BALB/c x AKR)F1 x AKR backcross mice. XC cell plaque assay was conducted on 1 cm terminal tail segments of six week-old mice. The mice were then observed for the development of leukemia, until the youngest was 600 days old. When in extremis the mice were sacrificed and autopsied. Of the mice observed, 33.1% developed leukemia during the 19 to 21 month observation period. Of these, 55.5% of the deaths occurred among mice which had shown detectable levels of MuLV in the XC cell plaque assay; 8.7% of the virus-negative mice died of leukemia. A hemagglutination method was used to determine the H-2 type of the mice. Leukemia developed in 42.8% of the H-2k/H-2k homozygotes, but in only 23.7% of the H-2k/H-2d heterozygotes. While both early MuLV presence and H-2 type can be correlated with the expression of leukemia, it appears that H-2 type has little influence on the early expression of the virus. No statistically significant difference in occurrence could be attributed to sex, even though sex is significant in the AKE parent.

112. EVALUATION OF T-LYMPHOCYTE REACTIVITY IN LEUKEMIA PRONE AKR MICE.

Biasi G, Collavo D, Biasi A, Chieco-Bianchi L
Inst. Pathol. Anat., Univ. Padova, Italy
Proc Fifth Int Symp Biol Characterization Hum Tumors, Bologna, April 4-6, 1973;261-265 1974

The function of lymphocytes from the peripheral blood and the spleen of normal preleukemic AKR mice was investigated and the T-lymphocyte reactivity of AKR mouse lymphocytes was compared with that of CBA mice, a strain which has the same H-2 histocompatibility locus but is characterized by a low incidence of spontaneous leukemia. No significant difference in the uptake of tritiated methyl-thymidine (1.5 microCi/ml for six hr) was observed between normal and preleukemic mice. In the preleukemic mice there was no decrease in the number of phytohemagglutinin-responsive lymphocytes/106 phytohemagglutinin-stimulated peripheral blood cells was significantly higher in CBA than in AKR mice. These results indicate that the number of phytohemagglutinin-responsive lymphocytes in peripheral blood and spleen cells is markedly lower in the AKR strain the

CBA strain. An impairment of immune reactivity seems to exist in the AKR mice.

113. RELATIONSHIP BETWEEN TUMOUR ANTIGEN AND HISTOCOMPATIBILITY ANTIGEN AT THE CELL SURFACE.

Meyer G, Berebbi M, Oth D
Unite de Cancerologie Experimentale U.119 de l'I.N.S.E.R.M. 27, bd Lei-Roure, F 13009 Marseille, France
Bull Cancer (Paris); 63(1):111-121 1976

The authors outline schematically the major histocompatibility complex as well as the relationship between tumour neo-antigen and pre-existing antigens at the cell surface. They note that these two antigenic systems are not independent and in particular, the major histocompatibility system (H-2) co-segregates with the tumour antigens. Considering the complexity of the H-2 system (a H-2 allele does not correspond to any single transplantation antigen but to a combination of several antigens units simultaneously present), the authors recall Boyse's hypothesis, revised by Haywood and McKhann, which proposes that tumour antigens are a rearrangement of H-2 substructures. Moreover, it is possible that the relationship between H-2 and resistance to cancer may be attributed directly to the action of genes controlling the immune response (Ir region) which could also intervene in recognition of these tumour antigens. Finally, recent results obtained seem to show that some tumour antigens crossreact with H-2 fractions. This fact prevents the mice bearing these types of alleles from being immunized against the cross-reacting tumours. If this result could be transposed to the human situation, it would explain the frequency of certain types of tumours in defined H-LA groups and could allow the prediction of high-risk groups.

V. ASSOCIATION BETWEEN CERTAIN HL-A HAPLOTYPES AND CANCER IN HUMANS

A. Association of Certain HL-A Alleles with Lympho-Hematopoietic Cancer

114. HL-A GENOTYPE OF PATIENTS WITH ACUTE LYMPHOBLASTIC LEUKAEMIA.

Singal DP, Naipaul N, Berry R, Pai MK, Zipursky A
Dept. Surg., McMaster U., Hamilton, Ontario, Canada
Humangenetik; 13(3):234-237 1971

A genetic study of ten patients with acute lymphoblastic leukemia is reported. All were typed for 21 HL-A specificities. Family analyses were used to determine the genotype of nine patients. All patients had different HL-A phenotypes; HL-A2 occurred in five patients, HL-A1 in four patients, and HL-A8 in four patients. Haplotype HL-A1,8 occurred in five of 18 instances. A slight increase in the incidence of antigens HL-A1 and HL-A8 was discovered phenotypically. The HL-A antigens present on cells of the leukemia patients were in accordance with the expectations from the family analyses. Thus, no loss of the HL-A antigens on lymphocytes could be detected in the present series.

115. THE HL-A SYSTEM IN LYMPHOBLASTIC LEUKAEMIA: A STUDY OF PATIENTS AND THEIR FAMILIES.

Lawler SD, Klouda PT, Hardisty RM, Till MM
Royal Marsden Hosp., London, England
Br J Haematol; 595-605 1971

To determine the relationship of the HL-A system to neoplastic blood disease the HL-A phenotypes of 58 Caucasian children with acute lymphoblastic leukemia, as well as the phenotypes of parents and sibs, were studied. It was found that the reaction frequencies of the internationally defined HL-A antigens, HL-A 1, 2, 3, 10, and 11 at the first sublocus and HL-A 5, 7, 8, 12, and 13 at the second sublocus, did not differ in the patients or in their sibs or fathers from normal controls. On the other hand, the mothers had a higher frequency of HL-A 11 antigen and a lower frequency of HL-A 13 antigen. FJH was the only antigen with a reaction frequency differing from the normal control value (3%) in patients (17%), fathers (13%), mothers (13%), and sibs (14%). The most important inference that could be drawn from this latter finding was that the FJH antigen, belonging to the second sublocus, was not preferentially coupled with any particular antigen at the first sublocus. It was also clear that the FJH antigen could be either paternally or maternally derived, that the segregation of the antigen was random, and that there was no deficiency of HL-A in the sibs. Finally, it was found that haplotypes occurring most frequently in the patients, parents, and sibs were also commonest in the control population. It is concluded that no particular HL-A phenotype or genotype is related to a predisposition to acute lymphoblastic leukemia; however, the high frequency of the FJH antigen needs to be investigated.

116. ACUTE CHILDHOOD LEUKAEMIA IN RELATION TO THE HL-A HUMAN TRANSPLANTATION GENES.

Walford RL, Finkelstein S, Neerhout R, Konrad P, Shanbrom E
U. California Los Angeles Med. Ctr.
Nature (Lond); 225(5231):461-462 1970

In a study of 10 families with 1 leukemic child each and other sibling(s) available for HL-A typing (23 children; 20 parents), all 10 leukemics were HL-A1 negative; 9/10 were phenotypically Merritt positive; and 7/10 were HL-A2 positive. The authors suggest that the presence of an HL-A 2 Merritt haplotype may indicate an increased susceptibility to leukemia, but not an absolute susceptibility since some healthy parents and siblings of the leukemics studied also possessed the HL-A 2 Merritt haplotype.

117. STUDY OF HL-A GENOTYPES IN A CASE OF FAMILIAL CHRONIC LYMPHOCYTIC LEUKAEMIA (CLL).

D[illegible]s-Marsalet Y, Hors J, Colombani J, Dausset J
Reg. Blood Transfus. Cent., Lille, France
Tissue Antigens; 4(5):441-443 1974

Blood groups and histocompatibility antigens (HL-A) were determined in 12 members of a family with four siblings suffering from chronic lymphocytic leukemia (CLL). The parental haplotypes were Da25, HL-A13/HL-A3, W5 and HL-A2, HL-A12/W32, W5. The same HL-A haplotype Da25 (W30 + W31), HL-A13 was shared by three of the diseased siblings. This case, together with a similar family study showing that three of five disease siblings shared two antigens (HL-A2 and W3), suggests a possible linkage between HL-A and susceptibility to CLL. The three hypotheses that could account for a dominant susceptibility outline are: the HL-A antigens (or products of very closely linked genes) are the receptors of the potential agent, these antigens share some structure with this agent which prevents immunization, and the dominant manifestation of an immune response gene.

118. HLA LINKED RESISTANCE FACTORS AND SURVIVAL IN ACUTE MYELOGENOUS LEUKEMIA.

Oliver RT, Pillai A, Klouda PT, Lawler SD
Dept. Medical Oncology, St. Bartholomew's Hosp. London, EC1, England
Cancer; 39(6):2337-2341 1977

The concept that HLA-linked immune response genes may influence survival of patients with malignant disease has been investigated by performing HLA typing on a series of 150 patients with acute myelogenous leukemia. Patients with A1 and B8 and/or A2 and B12 survive longer than patients without either of these pairs of antigens in association. However, after correction of the statistical probability for the number of A and B locus combinations theoretically possible, these differences are not statistically significant. Clarification of this must await independent analysis of other series and more direct approaches to the study of immune response genes in man. (Author abstract) (22 Refs)

119. HL-A ANTIGENS IN PATIENTS WITH HODGKIN'S DISEASE.

Maczynska-Rusiniak B, Zupanska B
Instytutu Hematologii, Warsaw, Poland
Acta Haematol Pol; 6(4):267-271 1975

The frequency of HL-A antigens was established in 46 patients with Hodgkin's disease and was compared with the incidence in the Polish population at large. Statistical analysis was carried out by the x square test. HL-A 1, 2, 3; W28 11, 9, 10; and HL-A5 7, 8, 12, 13, W5, W27, W10, W22, W15, and W17 antigens were determined. The most statistically significant difference between patients and healthy subjects was found with the HL-A5 antigen ($X^2 = 32.02$, $p = 0.005$), which appeared much more often in patients (43% vs 14.8%). The only other significant difference was found with W5, which was decreased in patients compared to healthy subjects (4.5% vs 13.5%; $X^2 = 7.08$, p less than 0.05). The explanation for increased HL-A antigen in Hodgkin's disease may lie in the similar construction of this antigen and determinants of oncogenic viruses or in the connection between genes controlling transplant antigens and those controlling the system's immunologic response.

120. INFLUENCE OF HLA GENES ON SURVIVAL IN HODGKIN'S DISEASE (MEETING ABSTRACT).

Osoba D, Falk J
Depts of Medicine and Surgery, University of Toronto, Toronto, Ontario, M4x 1k9
Proc Am Soc Clin Oncol; 17:279 1976

Seventy-nine patients with Hodgkin's disease were HLA typed before treatment and followed to determine if there is an association between HLA genes and survival in this disease. All survivors have been followed for a minimum of 30 months since diagnosis. The specificities AW19 and B5 were present either singly or together in 9 of the 14 patients (64%) dying from Hodgkin's disease within 42 months of diagnosis. This is an unexpectedly high frequency of these specificities. Thus, the entire cohort of 79 patients has been divided into 2 groups according to whether the patients were "positive" (24 patients) or "negative" (55 patients) for AW19 and/or B5. Both groups are similar with respect to age, sex and distribution of histological type of disease. There are significantly fewer patients with Stage IV disease in the "positive" group (8%) than in the "negative" group (29%). Nevertheless, the "positive" group has had a lower frequency of complete remissions after initial therapy (61% vs 83%) and a higher mortality (29% vs 7.3%; P=0.01) than the "negative" group during 30 months of observation. These results support previous suggestions (Lancet 2: 1118, 1971; J. Immunogenetics 1: 53, 1974) that genes in the HLA region play a role in the clinical course of Hodgkin's disease. (Author Abstract)

121. LEUKOCYTE ANTIGENS AND DISEASE. II. ASSOCIATION OF HL-A5 AND LYMPHOMAS.

Rege V, Patel R, Briggs WA
Rhode Island Hosp., Providence
Am J Clin Pathol; 58(1):14-16 1972

Forty-three healthy persons and 42 patients with biopsy-proven lymphoma (including 20 with Hodgkin's disease) were typed for eight histocompatibility antigens in a standard lymphocyte cytotoxicity test. The two groups differed only with respect to the HL-A5 antigen. Hodgkin's disease patients and patients with other lymphomas had about twice the incidence of positive reactions with anti-HL-A5 antisera (p less than 0.05) as did the normal controls. The association of an histocompatibility antigen with human lymphomas is analogous to the association of the H-2 mouse histocompatibility antigen with leukemia. Whether the increased susceptibility is due to a genetic factor or to a viral component as in the mouse, or to both, is unknown.

122. MULTIPLE CASES OF LYMPHOEPITHELIOMA AND BURKITT'S LYMPHOMA IN A CANADIAN FAMILY.

Joncas JH, Rioux E, Robitaille R, Wastiaux JP
Dept. Virology, Institut Armand Frappier, 531, Boulevard des Prairies, Case Postale 100, Ville de Laval, Quebec H7N 4Z3, Canada
Bibl Haematol; (43):224-226 1976

A family with multiple cases of lymphoepithelioma and Burkitt's lymphoma was studied to determine the implication of a genetic histocompatibility antigen (HL-A)-linked factor. The family consisted of the mother, 11 living children, and 2 living maternal cousins. The father, three additional siblings, and two additional maternal cousins had died prior to the study. One sibling and one cousin had died of abdominal Burkitt's lymphoma. Two siblings died of nasopharyngeal carcinoma (lymphoepithelioma), and the second cousin had an IgG plasmacytoma. Epstein-Barr virus (EBV) antibodies were measured, and HL-A typing was done by microcytotoxicity assay. The HL-A typing failed to disclose HL-A 2 or, in particular, HL-A Sin 2, which has been associated with nasopharyngeal lymphoepithelioma in Asia. The EBV antibody titers available in the two lymphoepitheliomas but not in the Burkitt's lymphomas were markedly elevated. The occurrence of a plasmacytoma in the first-degree cousin suggests the possibility of a predisposing B cell dysfunction in the development of these tumors. In certain genetically predisposed hosts, B cell dysfunction could possibly lead to B cell lymphomas.

123. HLA HAPLOTYPES IN A GENETIC ISOLATE IN NEWFOUNDLAND. A POPULATION SHOWING 8% HOMOZYGOSITY AND A FAMILIAL AGGREGATE OF LYMPHOMA AND IMMUNODEFICIENCY CASES.

Larsen B, Barnard JM, Buehler SK, Marshall WH
Faculty of Medicine, Memorial University of Newfoundland, St. John's, Newfoundland, AIC 587 Canada
Tissue Antigens; 8(3):207-215 1976

HLA typing was performed on 384 individuals of an isolated population of 1,500 people with a familial aggregate of lymphoma and immunodeficiency cases. Eighty-five % of the total population were descendants of the founding couple. First cousin marriages were common. There was a three-fold or higher increase of the following haplotypes as compared to the frequencies in Sheffield: HLA-A28, Bw35, HLA-A28, B18, HLA-A10, B18, HLA-A2, B18, HLA-A11, Bw40 and HLA-A11, B7. The frequency of HLA-A1, B8 was low (5.4%). The most common genotype was HLA-A2, B12/A2, B12 followed by HLA-A2, B12/A28, Bw35. We found 30 HLA homozygous individuals, of these 15 were HLA-A2, B12/A2, B12. There were two possible HLA cross-overs which may be confirmed and three postulated cross-overs which can never be confirmed as one or both parents of the individuals in question are deceased. Some of the haplotypes could be traced back to the first, second and third generations, ie, to the first half of the nineteenth century. No single haplotype or antigen was shared by the patients. (Author Abstract)

124. HL-A ANTIGEN FREQUENCIES IN MYELOMA.

Mason DY, Cullen P

Radcliffe Infirmary, Woodstock Road, Oxford, OX2 6HE England

Tissue Antigens; 5(4):238-245 1975

The frequency of HL-A antigens was determined in 63 myeloma patients and in 83 normal subjects. The patients consisted of 32 males and 31 females, with an average age of 60. The distribution of paraprotein types was IgG: 37, IgA: 15, light chain disease: 10, and IgD: 1. The ages of the controls range from 18-64. Tissue typing was performed using a microlymphocytotoxicity test with lymphocytes separated from defibrinated venous blood by centrifugation on Triosil/Ficoll. The HL-A antisera had been selected within the London Transplant Group for typing renal homograft donors and recipients. Eighteen well-defined antigens were studied: HL-A1, 2, 3, 9, 10, 11 and 28 at the first locus, and HL-A5, 7, 8, 12, 13, 14, 17, 27, W10, W15, and W22 at the second locus. All patients and normal subjects were also tested with antisera detecting the W5 and/or W18 antigens. In addition, 42 of the patients and 75 of the controls were tested for the W29 and Da25 components of the W19 antigenic group. The 4c antigen group was taken as that detectable by antisera reacting with both Hl-A5 and W5/W18 antigens. Only the HL-A5 antigen showed a significant difference in frequency between the two groups; it showed a frequency of 15.9% among patients, and 3.6% among controls. Group 4c antigens were also common among patients (31.7%) than controls (15.6%). Whether this difference can be accounted for by the presence of non-HL-A specificities is uncertain.

B. Association of Certain HL-A Alleles with Solid Tumors

125. IMMUNOGENETIC ASPECTS OF NASOPHARYNGEAL CARCINOMA: I. DIFFERENCES IN HL-A ANTIGEN PROFILES BETWEEN PATIENTS AND CONTROL GROUPS.

Simons MJ, Wee GB, Day NE, Morris PJ, Shanmugaratnam K, De-The GB

WHO Immunol. Res. Training Ctr., Dept. Path., U. Singapore

Int J Cancer; 13(1):122-134 1974

Preliminary analysis of HL-A antigen patterns in 393 subjects showed a higher frequency of undetectable second locus antigens (particularly HL-A13 and W10) in 28 Singapore Chinese patients with nasopharyngeal carcinoma (NPC) compared with 175 Chinese, 104 Malays, and 86 Indians without NPC. The same phenomenon was demonstrated in a study of 136 NPC patients (74 Singapore Chinese, 14 Hong Kong Chinese, 28 Malaysian Chinese, 12 Malays, 7 Kadazans, and 1 Dyak) and 61 Singapore Chinese without NPC. In this study, the significance of the second locus difference between Chinese with NPC and Chinese controls was increased 10-fold. Additionally, the frequency of HL-A2 was significantly higher among the NPC patients. Both the elevation of HL-A2 frequency and the deficit of detectable second locus antigens remained significant after application of the most conservative correction factor for the number of antigens investigated. The increase in HL-A2 was represented by the first locus component in Chinese NPC patients and by the second locus component in non-Chinese NPC patients. The lack of second locus antigens could be either a result of NPC or a genetic marker of susceptibility to the development of the malignancy.

126. IMMUNOGENETIC ASPECTS OF NASOPHARYNGEAL CARCINOMA (NPC) III. HL-A TYPE AS A GENETIC MARKER OF NPC PREDISPOSITION TO TEST THE HYPOTHESIS THAT EPSTEIN-BARR VIRUS IS AN ETIOLOGICAL FACTOR IN NPC. (PP. 249-258)

Simons MJ, Wee GB, Chan SH, Shanmugaratnam K, Day NE, De-The G

Oncogenesis and Herpesviruses II Part 2, Epidemiology, Host Response and Control Proceedings of a Symposium held in Nurember, Federal Republic of Germany, 14-16 October 1974. De-The G, Epstein MA, zur Hausen H, Davis W, ed. International Agency for Research on Cancer, Lyon, France, IARC Scientific Publications No. 11, 1975.

HL-A typing of 144 nasopharyngeal carcinoma (NPC) patients and 236 controls from China revealed an increased frequency of first locus HL-A2 and an deficit of antigens at the second locus in the NPC patients. HL-A2 and the second locus blank appeared to act together (HL-A2 blank haplotype) in determining NPC risk in highest-risk Cantonese, whereas in relatively lower-risk non-Cantonese Chinese (Hokkiens, Teochews) they appeared to act independently. That is, only the blank had an increased frequency in Malay NPC patients. The strength of the HL-A association with NPC reflected the thirty- to fiftyfold difference in incidence between highest-risk Cantonese and lowest-risk Indians. In Singapore, HL-A segregation patterns in families of nine Chinese NPC patients confirmed that the blank was a genetic phenomenon. A new second locus antigen, Singapore-2, occurs more frequently in NPC patients. The HL-A association with NPC probably reflects the existence of disease-susceptibility (DS) genes in linkage disequilibrium with alleles of the HL-A loci. NPC-DS genes may determine differences in immune responsiveness to environmental agents and thereby determine differences in NPC incidence. If the known altered immune responsiveness of NPC patients to Epstein-Barr virus (EBV) reflects the function of DS genes linked to the high NPC risk HL-A type, then the hypothesis that EBV has an etiological role in NPC can be tested by prospective studies.

127. NASOPHARYNGEAL CARCINOMA. V. IMMUNOGENETIC STUDIES OF SOUTHEAST ASIAN ETHNIC GROUPS WITH HIGH AND LOW RISK FOR THE TUMOR.

Simons MJ, Day NE, Wee GB, Shanmugaratnam K, Ho HC, Wong SH, Ti TK, Yong NK, Darmalingam S, de-The G

WHO Immunology Res. Training Ctr., Singapore, Malaysia

Cancer Res; 34(5):1192-1195 1974

One hundred forty-four Chinese patients with nasopharyngeal carcinoma (NPC), 148 normal Chinese, and 88 patients, suspected on clinical grounds of having NPC but in whom no evidence of cancer was seen in histological preparations of biopsy tissue and who were therefore presumed to be free of NPC, were studied for HL-A antigen patterns. The antisera used identified up to eight lst-locus and up to 14 2nd-locus specificities. The two major findings in the NPC patients were a significantly increased frequency of HL-A2 and a nonspecific deficit of 2nd-locus antigens. The proportion of patients with the high-risk characteristic (HL-A2; less than two 2nd-locus antigens) versus low-risk individuals (lacking HL-A2; two 2nd-locus antigens) differed between the NPC patients and the comparison groups to a high order of statistical significance (X2 = 16.15; p = 0.00006; relative risk = 4.5).

128. THE RELATIONSHIP BETWEEN NASOPHARYNGEAL CARCINOMA AND THE HL-A SYSTEM AMONG TUNISIANS.

Betuel H, Camoun M, Colombani J, Day NE, Ellouz R, de-The G

Centre de Transfusion Sanguine de Lyon, Beynost, 01700 Miribel, France

Int J Cancer; 16(2):249-254 1975

A study was conducted to determine whether or not a similar relation of HL-A antigens and increased risk of nasopharyngeal carcinoma (NPC) found among the Chinese (Singapore, 1975) is also present in Tunisians. Blood samples were obtained from 109 NPC patients, in 39 patients with other forms of cancer, and in 45 intermittent noncancerous controls. More than two monospecific antisera were used to detect each HL-A antigen. No significant differences were observed between the NPC and nonNPC patients for any specific antigen; however, there was a slightly higher frequency of A2 among the nonNPC cases than among the NPC cases. At the first H1-A locus, no association was found between A2 and NPC, in contrast to the Singapore results. Thus, the results were similar to those found in Singapore, but the effect was less marked and not statistically significant. The authors conclude that though the results are close to what might be expected for an association of increased NPC risk and the second locus blank, the study would have to be continued for several years to give statistically significant results.

129. NASOPHARYNGEAL CARCINOMA: PRESENT STATUS OF KNOWLEDGE.

Henderson BE

U. Southern California Sch. Med., Los Angeles

Cancer Res; 34(5):1187-1188 1974

The possible role of genetic factors in the etiology of nasopharyngeal carcinoma (NPC) is indicated by the fact that a relatively high rate of NPC is found among the Southern Chinese, a lower rate is present among Chinese from the more northern provinces, and the disease is relatively rare in Japan and India. The greater the admixture of Chinese blood in an ethnic group, the more likely it is that the NPC incidence rates will be elevated. Although NPC rates remain high in Chinese who immigrate to California, successive generations of Chinese born in the United States appear to be at decreasing risk. An increased frequency of the histocompatibility antigen HL-A2 may be a marker of genetic susceptibility to NPC in the Chinese population. Most environmental variables which have been suggested to play a role in the etiology of NPC have not been shown to be linked with increased risk of the disease. However, virtually 100% of NPC cases have elevated antibody titers to Epstein-Barr virus (EBV) regardless of the racial or geographical location of the cases. Whether EBV is a necessary cofactor for the development of NPC or a "passenger" virus remains unsolved. Although additional epidemiological evidence and age curve data indicate that some environmental factor is involved in the etiology of NPC, additional research is needed to confirm this hypothesis. Studies among populations with a lower rate of NPC are suggested.

130. LYMPHOCYTE FUNCTION AND HL-A ANTIGEN FREQUENCY IN GYNECOLOGIC SQUAMOUS CANCER: A PRELIMINARY REPORT.

Twomey PL, Rogentine GN, Chretien PB

Natl. Inst. Health, Bethesda, Md.

Int Surg; 59(9):468-472 1974

Cellular immune competence in 51 patients with gynecologic neoplasms was measured by quantitating lymphocyte response to phytohemagglutinin. The 51 patients included 31 with squamous cell carcinoma of the cervix, 8 with squamous cell carcinoma of the vulva or lower vagina, and 12 with adenocarcinoma of the uterus. Genetic make-up in 43 cancer patients was characterized by determining human lymphocyte antigen (HL-A) types. Thirty-eight of the 39 patients with squamous cell cancers had low lymphocyte reactivity (38%), whereas no abnormality was found in patients with adenocarcinoma of the same organ. The response in squamous cancer patients differed significantly (p less than 0.01) from that of controls and (p less than 0.025) that of the adenocarcinoma patients. Among squamous carcinoma patients, the frequency of HL-A8 was nearly 2 1/2 times higher than in control. This antigen has previously been linked to benign diseases with altered immune fraction and to trophoblastic malignancy. Abnormal HL-A antigen frequencies and immune defects in women with gynecologic squamous cell cancers may be important clues to the causes of these cancers and guides to therapy.

131. HL-A ANTIGENS AND BREAST CANCER.

De Jong-Bakker M, Cleton FJ, D'Amaro J, Keuning JJ, Van Rood JJ

Netherlands Cancer Inst., Amsterdam

Eur J Cancer; 10(9):555-558 1974

Frequencies of HL-A antigens in 200 patients with breast cancer were compared to those in 200 matched controls to determine the relationship of the antigens to the disease. Following HL-A typing, correlations between antigen frequency and clinical data were calculated. No correlation was found between HL-A antigens and breast cancer, but an increased frequency of the combination HL-A 9-HL-A 27 was observed. A study of breast cancer families is suggested to search for linkage of genetic markers with breast cancer and to determine the possibility of HL-A haplotypes.

132. HL-A ANTIGENS AND THE CHORIOCARCINOMA.

Ivaskova E, Jakoubkova J, Zavadil M, Schneid V, Koldovsky P, Ivanyi P

Inst. Exp. Biol. Genet., Czech. Acad. Sci., Prague

Folia Biol (Praha); 14(5):398-401 1968

The presence of 5 antigens of the major human histocompatibility system (HL-A) was studied (using specific antisera) in the sera of 15 women with trophoblastic tumors (choriocarcinoma, "proliferating mole" or hydatidiform mole), their husbands, and 20 normal control women. A significant correlation was seen between the presence of the tumors and the absence of antigen 1 of the HL-A system; sera from 12/15 tumor patients were negative, whereas sera from 11/15 of the husbands and 8/20 control women were positive for antigen 1. Another significant difference between the patients and the control groups was seen in another study using 87-122 unclassified cytotoxic sera; sera from the husbands and the control women reacted with 52-53% of these cytotoxic sera, whereas sera from the patients reacted with only 39% of these sera. The sera of 12/13 patients examined showed cytotoxic antibodies against their husbands' WBC. It is suggested that the presence of HL-A alleles, with relatively few antigenic specificities (because of aberrations of the chromosomes carrying the gene(s) determining the HL-A system) is the probable condition for the occurrence of trophoblastic tumors. Under these conditions, an immune reaction to the husbands' HL-A antigens may occur in some patients, enhancing the growth and possibly causing the malignancy of the allograft. Incompatibility for antigen 1 or its alternative antigens may play a decisive role under these circumstances.

133. HL-A ANTIGENS AND CHORIOCARCINOMA.

Rudolph RH, Thomas ED

U. Washington Sch. Med., U.S. Public Hlth. Service Hosp., Seattle

Lancet; 2(7721):408-409 1971

A woman who had given birth to a full-term, live infant and who had developed disseminated choriocarcinoma during that pregnancy was studied with respect to histocompatibility factors using both leukocyte sero-typing and one-way mixed-leukocyte culture. Differences in HL-A antigens (leukocyte antigens controlled by genes at the HL-A locus) were found between the infant and his mother with both techniques, disproving the hypothesis that the survival and dissemination of post-gestational choriocarcinoma are dependent upon histocompatibility between the malignant graft and the maternal host. Alternative mechanisms, possibly those involving serum blocking or enhancing factors, which permit tumor-graft survival must be sought.

C. General Studies on the Association of HL-A Antigens with Human Disease

134. HUMAN IA-TYPE ALLOANTIGENS: METHODS OF DETECTION, ASPECTS OF CHEMISTRY AND BIOLOGY, MARKERS FOR DISEASE STATES.

Wernet P

Dept. Medicine II, Univ. Tubingen, D-74 Tubingen, W. Germany

Transplant Rev; 30:271-298 1976

Developments concerning the specific human B lymphocyte alloantigens linked to histocompatibility (HLA) are reviewed with special emphasis on detection and classification and some molecular aspects. The biological action of human Ia antigens is discussed, and the utility of a new system dealing with immunopathology is illustrated. A variety of methods are currently in use for detection: immunofluorescence, cytotoxicity (including tandem typing and automated tandem cytotoxicity using fluorochrome), and radioimmunoassay serotyping (radioscreening for anti-Ia antibodies in pregnancy sera and direct radioimmunotyping for Ia determinants on normal purified B lymphocytes). The principal method used to study the molecular structure of lymphocyte antigens is lactoperoxidase-catalyzed surface radioiodinization of lymphocytes and electrophoretic analysis of specific radioactive immunoprecipitates. Clearly differential patterns of positive and negative reactions obtained with individual sera have shown that a large number of different alloantigens as well as overlapping specificities are involved. Certain sera have specificity patterns corresponding to HLA-D typing. A study in a cousin-marriage family with a double crossover between HLA-B and HLA-D is presented to demonstrate successful serotyping of mixed lymphocyte culture determinants with B cell alloantibodies. There is probably a large number of specific B cell alloantigens controlled by genes linked closely to those controlling the expression of HLA alloantigens. Antisera directed against only H-2D have no effect on the primary or secondary responses to SRBC, but anti-Ia antisera do inhibit both types of immune reactivity. B cell alloantibodies can inhibit secondary immune responses of human purified B cells when incubated prior to antigen exposure. Several lines of evidence have indicated that human B cell alloantigens are not restricted to lymphocytes. With use of a variety of B cell alloantisera, 13/17 acute lymphoblastic leukemia cells showed the presence of Ia type antigens; none of the usual surface markers for T and B cells was found. A 100% association of the Ia specificity (which is DW2-associated) has been demonstrated in more than 100 multiple sclerosis patients.

135. MOLECULAR ASSOCIATION OF HISTOCOMPATIBILITY AND TUMOUR ANTIGENS ON THE MEMBRANE OF MALIGNANT TUMOUR CELLS FROM CHILDREN (MEETING ABSTRACT).

O'Keefe DE, Wilson FC, Jose DG

Royal Children's Hosp. Res. Foundation, Melbourne, Victoria, Australia

Clin Exp Pharmacol Physiol; 3(3):276-277 1976

Structural changes occur in the plasma membrane of cells following mutation to malignancy. These foreign structures elicit immunological recognition and responses in the tumor-bearing patient. Identification and purification of antigenic components of tumor cell membranes are fundamental to analysis, diagnosis and possible vaccination for cancer. Soluble membrane extracts were prepared by spontaneous metabolic release and by extraction using hypertonic salt solutions or non-ionic detergent. Tumor cells and equivalent normal tissue were obtained from children with acute leukemia, osteosarcoma, neuroblastoma, rhabdomyosarcoma, Wilm's tumour and astrocytoma. Membrane analyses were performed on radio-iodinated membrane preparations by SDS-polyacrylamide gel electrophoresis, iso-electric focusing, immuno-coprecipitation and affinity chromatography. Functional characteristics were determined on membrane fractions purified by gel filtration and affinity chromatography. Membrane components common to all tumor types formed sharply defined peaks of glycoprotein in 11,000 and 50,000 daltons area on gel electrophoresis, with variable amounts of material in intermediate ranges. Autologous tumor antigens, defined by both serum and cells from the patient in remission were found in both high and low molecular weight fractions with additional activity on variable peaks in the 30-35,000 daltons range. These major glycoprotein fractions also contained antigen determinants of both first and second HL-A loci and for mixed leucocyte reactivity. Autologous tumor antigens and major histocompatibility antigens could not be separated by specific coprecipitation with HL-A antisera. Quantitative absorption of HL-A antisera suggested an inverse relationship between antigenic strength of histocompatibility and tumor antigens at the membrane. The association of normal histocompatibility and tumor antigens at the molecular level suggests a fundamental role for these membrane structures in regulation of cell-cell interaction and growth.

136. THE ASSOCIATION OF THE HL-A ANTIGENS WITH DISEASES.

Dausset J, Degos L, Hobs J

Lab. Immunol. Hematol., Univ. Paris, France

Clin Immunol Immunopathol; 3(1):127-149 1974

The possible mechanisms of association between the HL-A antigens and various neoplastic and non-neoplastic diseases are reviewed. Experimental data indicate some correlation between a susceptibility to oncogenic viruses and the immune response and the H-2 system in mice; the H-2 system corresponds to the HL-A system in man. However, in man there is no clear association between any of the HL-A antigens and acute lymphocytic or other types of leukemia. There is a close association between HL-A8 and gluten enteropathy, dermatitis herpetiform and systematic Lupus Erythematosus, while ankylosing spondylitis, psoriatica arthritis, Reiter's disease, and Yersinia arthritis are associated with W27, and psoriasis and pemphigus are associated with HL-A13. These diseases, as well as several others which are associated with various HL-A antigens must be distinguished from those, such as ragweed pollen allergy, in which there is an apparent link between the HL-A system and an abnormal gene. With regard to the diseases which are associated with a given HL-A antigen, it is possible that the HL-A products are themselves responsible for the association with disease; given the well-established presence of Ir genes in mice and guinea pigs, it is also possible that similar genes exist in man. The fact that Ir genes are probably present in the HL-A complex permits the prediction that heterozygotes in this region will be able to develop a larger spectrum of antibodies and will accordingly enjoy a biological advantage; there is some evidence that homozygotes are more susceptible to disease and especially to cancer. The HL-A complex may well find its ultimate mission in its relationship with selection, as well as in transplantation.

137. HL-A AND CANCER. (PP. 107-111)

Govaerts A

Clinical Tumor Immunology. Wybran J, Staquet MJ, ed. Oxford, Pergamon Press, 1976.

The association between HL-A antigens and diseases is discussed. A total of nearly 600 patients with breast cancer has been typed in different laboratories; the combined analysis did not show any association with HL-A antigens. Malignant melanoma was investigated in 350 patients without any correlation between single HL-A antigens and the disease. However, the W30-HL-A12 haplotype appeared much more frequently than in normal subjects. Lymphomas displayed the HL-A 9-5 haplotype; whereas, kidney cancers had the haplotype 2-12 and stomach cancer the haplotype 3-7. There was no difference between 40 acute lymphoblastic leukemias and the normal population in typing for 12 antigens at the first locus and 16 at the second. Either a weak association or no association was observed between HL-A and chronic lymphoid leukemia, chronic myeloid leukemia, Hodgkin's disease, or Burkitt's disease. It is hypothesized that there may be a linkage disequilibrium between some Ir and HL-A genes or a cross-antigenicity between some HL-A determinants and microorganisms. (13 refs)

138. HL-A ANTIGENS AND SUSCEPTIBILITY TO DISEASE.

Bertrams J

Elisabeth-Krankenhaus, 4300 Essen, Moltkestr. 61, West Germany

Dtsch Med Wochenschr; 101(5):178-184 1976

HL-A (histocompatibility) antigens and disease susceptibility, the HL-A supergene, the serologically defined loci on chromosome 6 and their phenotypic frequency, and HL-A-linked pathology are topically reviewed. Known HL-A antigens for Hodgkin's disease, acute lymphocytic leukemia, and breast carcinoma are compiled from the literature; most results are equivocal. Uterine, cervical, and ovarian carcinoma, chronic lymphocytic and chronic myelogenous leukemia, various sarcoma types, melanoma, retinoblastoma, nasopharyngeal carcinoma, and Burkitt's lymphoma show no association with single HL-A antigens. Various explanations of the correlation between pathogenesis and determinants of the histocompatibility complex are explored. (47 refs)

139. HISTOCOMPATIBILITY IN CANCER. (PP. 76-86)

Takasugi M, Krain L, Terasaki PI

Cancer Genetics. Edited by HT Lynch Springfield IL, Charles C Thomas, 1976.

The discovery of and early experiments on histocompatibility antigens are reviewed, as are studies on the genetics of resistance and susceptibility to viral carcinogenesis and immunological responsiveness. Most animal models appear to possess a single major genetic histocompatibility system. Serological and biochemical studies have emphasized the homologies between the H-2 (mouse) and HL-A (human histocompatibility locus) systems. The consistent association between histocompatibility antigens and susceptibility to cancer in mouse systems suggests important biological relationships, with parallels to be sought in man. Their proposed mechanisms fall into two categories: (a) a gene-controlling responsiveness linked to histocompatibility; and (b) unresponsiveness as a result of cross-reaction with one's own antigens. The response in F1 to polypeptide antigens and gross-virus leukemogenesis indicates that genetic control is other than tolerance to one's own antigens. The many studies of the connection between Hodgkin's disease and HL-A indicate some association of antigens in the HL-A5, W5, W8, 4c complex with Hodgkin's disease. In a large-scale study, patients with cancer of the prostate, stomach, endometrium, and ovary and with melanoma showed essentially identical HL-A frequencies as the control population. Patients with cancer of the bladder, breast, lung, colon, and rectum and patients with lymphomas and lymphosarcoma exhibited deviations from control levels for only 1 of 23 specificities at a significant level. Among patients with carcinoma of the cervix, HL-A1 was 35% and HL-A12 was 32% compared to 27% and 24% respectively, in normal subjects. HL-A9 was 15% in these patients compared to 21% for normals. In a series of 321 patients with Hodgkin's disease, W5 and W18 were elevated at the expense of HL-A7. The potential value of HL-A antigens as possible markers for cancer susceptibility or resistance genes emerges best from family studies, many of which will have to be done across generations.

140. RELEVANCE OF MAJOR HISTOCOMPATIBILITY DETERMINANTS IN CLINICAL BONE MARROW TRANSPLANTATION.

Hansen JA, O'Reilly RJ, Good RA, Dupont B

Memorial Sloan-Kettering Cancer Center, 1275 York Ave., New York, NY 10021

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Grafting between HL-A-identical siblings has made possible many recent advances in clinical bone marrow transplantation. Bone marrow transplantation between HL-A-nonidentical individuals, however, has invariably resulted in fatal graft-versus-host reactions (GVHR). Marrow transplantation in aplastic anemia and leukemia patients is very difficult, and the complications are more severe than those occurring after transplants in infants with severe combined immunodeficiency disease (SCID). Despite pretreatment with cyclophosphamide (CP) alone or a combination of CP, procarbazine, and antithymocyte serum, the graft rejection rate is 25%-30% for aplastic anemia patients receiving HL-A-identical sibling marrow. Marrow cells from a family donor differing only with respect to a single HL-A haplotype will regularly produce fatal GVHR in infants with SCID. However, successful grafting has been achieved by matching the patient with the donor on the basis of relative nonreactivity in the mixed lymphocyte culture (MLC) test. Therefore, matching for MLC is emphasized in selecting marrow donors. Although the MLC test reflects stimulation by HL-A determinants and the response phase of immunocompetent cells, the cell-mediated lympholysis test reflects both a response and effector phase of cell-mediated immunity. The HL-A region is becoming seemingly more complex as new determinants and biologic effects are realized. The HL-A D region may be associated with closely linked genes controlling B-cell alloantigens and CML determinants, in addition to the alleles that control MLC-stimulating determinants. Although genotypic identity with the HL-A complex may not necessarily prevent GVHR, alteration of the expression of histocompatibility determinants by chemotherapy, radiation, or virus infection may potentiate these reactions. (39 refs)

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